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NEUROLOGICAL COMPLICATIONS OF SERUM SICKNESS WITH SPECIAL REFERENCE TO THE EAR.*

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In curative and preventive treatment, foreign serums administered parenterally may affect adversely both the somatic and the sympathetic nervous systems. It has long been observed that numerous syndromes follow their use, and it appears that the brain, spinal cord, meninges, spinal roots and cranial and spinal peripheral nerves may be involved in varying degree. Usually the symptoms subside, but not infrequently there may be residual damage. The etiological, pathological and clinical aspects of the neurological complications of this therapy and the prognostic considerations as well have gradually become more and more widely recognized.

Since 1908, when cases of this disease were first described,[1] reports have accumulated in the literature of France, England, Germany and the United States, and an occasional report has come from Belgium, Italy, Switzerland, Czechoslovakia, Denmark, Poland, Rumania and Japan.[2,3] In his review of the literature, Bennett[2] mentioned that the greatest number of cases have been reported from France, the report of Lhermitte[4] in 1919 stimulating interest there, and he added that Richardson[5] in 1917 reported the first case in the literature of the United States. General reviews of the subject have been presented during the last 15 years in the French, English, German and American literature.[2,6,7,8,9,10,11,12,13,14]

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ETIOLOGY.

It has for some years been recognized that an allergic or anaphylactic reaction of the body to a foreign serum characterizes the disorder, and it has also been established that the specific antitoxins in the serum have no etiological significance. The administration of serums for the prevention or treatment of tetanus. diphtheria, scarlet fever, tuberculosis and infections caused by gangrene and by the pneumococcus, gonococcus, meningococcus and streptococcus may cause reactions that present identical clinical pictures.

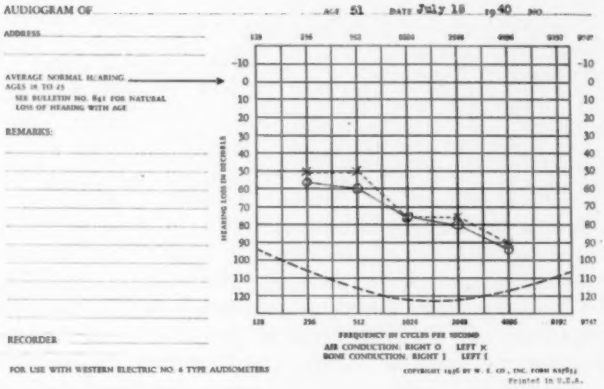
Most frequently serum sickness occurs following the administration of antitetanus serum, a serum given less often to children than to adults, in whom neurological complications are more likely to occur. Bennett[2] observed that in more than 50 per cent of the reported cases the condition followed the prophylactic use of tetanus antitoxin, although it was produced by practically all types of horse serum antitoxin. He noted also that the average age of the patients in the reported cases was 26 years and that women were less frequently affected than men. Doubtless the disease occurs oftener in man because of the relatively greater frequency of accidents among them, with the consequent necessity of greater use of antitetanus serum. It is believed that the youth of the patient to whom diphtheria antitoxin is usually given accounts for the relative rarity of cases of serum sickness following its use, for children are comparatively immune to this complication.

PATHOLOGY.

According to one of the two theories of serum disease, the primary site of this disorder is the blood vessels; according to the other, it is the cells. Experimental, postmortem and laboratory evidence and clinical manifestations indicate that the pathological process associated with serum sickness includes intense vasodilatation and edema of the skin, muscles, bursae and joint tissues. It is reasonable to conclude that the neuropathological changes parallel those occurring elsewhere in the body and, as described by Kraus and Chaney,[3] "consist of a primary disorder of the blood vessels, causing nutritive impairment of the tissues of the nervous system and interfering temporarily as a rule with the activity of the nerve

fibres and cells, but occasionally causing cell death and parenchymal necrosis." These authors noted that a vascular disorder also produces the meningeal picture and excess of spinal fluid, and they concluded that "an edematous process may occur in the perineural sheaths, or the nutrition of the roots or nerves may be interfered with by impairment in the blood supply, thus causing various radicular and neural syndromes."

As with the exact cause of serum disease, the exact pathological process associated with the occurrence and localization of serum neuritis is not established.[2] Early authors[5] ascribed it to involvement of the nerves by direct toxic action of the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serum, and others[15,16] to the effect of protein toxins on muscles innervated by nerves of low or medium chronaxia, but many writers[3,9,12,13,17] attributed it to perineural urticaria and edema producing a compressive or ischemic paralysis of nerve trunks or peripheral nerves. Of most frequent occurrence in reported cases of serum neuritis is the involvement of the suprascapular nerve combined at times with involvement of the axillary nerve or long thoracic nerve, all from the Vth, VIth and VIIth cervical nerves. Involvement, either unilaterally or bilaterally, of the upper brachial nerves with the resulting Erb-Duchenne scapulohumeral paralysis remains unexplained. The explanation of this localization must lie in a localized anatomical condition that resembles the compressive neuritis of Bell's facial paralysis or the compression of

the anterior scalene muscle that accompanies neuritis of the cervical rib.[2]

Since the Vth and VIth cervical nerves are most frequently affected to the greatest degree in cases of paralysis of the brachial plexus due to serum sickness, it may be that the anatomical explanation of their selection lies in their position. They come from the vertebral foramina higher in the neck than do the other nerves. Consequently, for them the distance to be traversed to the axilla is greater than for the other nerves of the plexus. Also, in their journey they must pass through the dense, tough fasciae of the neck, between the anterior and middle scalene muscles, and in close proximity to the other muscles of the neck and clavicle. Edema of the neural sheaths as a response to the serum sickness, which is probably the cause of the paralysis, of necessity causes enlargement of the nerve. Since the Vth and VIth cervical nerves must follow a longer course close to many structures, it is likely then that the swelling of these nerves and the resulting pressure against these structures bring about a pressure ischemia of the nerves with consequent paralysis.

Although a very frequent site of neural disorder associated with serum sickness is the Vth and VIth cervical nerves, there are numerous other sites of involvement. In addition to the manifestations occurring commonly in the peripheral nerves, there may be meningeal, spinal and cerebral lesions. In the cerebral form of the complication the cranial nerves may or may not be involved. The syndromes produced vary widely.

GENERAL COURSE AND SPECIAL SYNDROMES.

In the series of cases reported by Doyle,[14] the average interval between the first injection of serum and the onset of serum disease was 6.8 days. This author observed that it is difficult to determine the average interval between the onset of the sickness and the appearance of neurological complications, but he added that it was 2.1 days for the cases in his series in which this information was recorded.

The symptoms associated with serum sickness are usually pain, moderate or high fever and urticaria, which may be general or local. In addition, headache, nausea and vomiting may occur. The lymph nodes may become enlarged and pain-

ful; there may be swelling and an elevation of the temperature of one or more joints. Severe neuralgia in one or several extremities may be present. The site of injection bears no relation to the site of involvement of the nervous system. The numerous syndromes that occur may be classified as spinal, radicular, neural and cerebral.

As a rule the spinal, radicular and neural syndromes are easily established, but combinations of these syndromes are frequently observed. As previously mentioned, the most characteristic picture is that of Erb-Duchenne paralysis, involving the muscles supplied by the Vth and VIth cervical roots. Features of the cerebral syndrome are meningeal irritation, choked disks, alexia, hemianopsia, aphasia and hemiplegia. There is a great increase in spinal fluid pressure and cellular reaction is slight. Paralysis of the cranial nerves or a bulbar syndrome, with or without tetraplegia, may occur. Isolated involvement of the axillary, ulnar, median, radial, external popliteal or entire sciatic nerve has been reported. A single muscle, as, for example, the serratus anterior or the deltoid muscle, may be affected. Optic neuritis was mentioned by Kraus and Chaney[3] as one disturbance of the cranial nerves, and they observed that the oculomotor nerves and the VIIth, IXth, Xth and XIth nerves may be affected, either alone or in combination with other syndromes. Coons and Kirk[18] in an article entitled, "Serum Disease with Special Reference to Polyneuritis," referred to patients with optic neuritis, an edematous retina and swollen disk, ptosis, strabismus, diplopia, nystagmus, facial paralysis, husky voice, earache, tinnitus, deafness, hemiplegia, paraplegia, pseudotabes and other sensory and motor disturbances. Bedell[19] also mentioned an edematous retina, and Brown[20] referred to a swollen disk.

PROGNOSIS.

In general, the prognosis is good. Recovery usually occurs despite the severity of the symptoms, although complete restoration may require months. Residual paralysis or weakness of one or more muscles is occasionally observed; it may be partial or complete.

TREATMENT.

The treatment for neurological complications is in the acute stages identical with that of serum sickness in general. The

use of such drugs as atropine and epinephrine is indicated. The accepted methods of dehydration are employed when the cerebral syndrome is present, as it appears to be characterized by a considerable degree of edema. The intake of fluids is restricted, and retention enemas of magnesium sulfate are given. Drainage by repeated spinal puncture is carried out. Large doses of caffeine and intravenous injections of dextrose are given. The use of dextrose and caffeine in the treatment of cases of this type was suggested by Kennedy.[12] Electrical therapy and massage are beneficial in cases of muscular paralysis and wasting.[3]

AURAL MANIFESTATIONS.

Involvement of the cranial nerves, particularly the VIIIth nerve, as a complication of serum sickness occurs infrequently, but its very rarity emphasizes the importance of reporting such cases. Of the 49 cases of neurological complication resulting from this disease reviewed by Doyle,[14] 34 occurred following the use of tetanus antitoxin. In the majority of these cases the brachial plexus was involved, and in a few there was involvement of the optic nerve. In 1935, Amberg and Hewitt[21] reported the case of a boy, age 17 years, who had been given 120,000 units of tetanus antitoxin in the treatment of tetanus with resulting inner ear deafness. The deafness appeared to be total and did not improve. The following year, Cutter[22] reported a similar case with improvement. In this case a boy, age 14 years, was given 125,000 units of tetanus antitoxin in the treatment of tetanus. One week later, loss of hearing was perceptible; the deafness progressed gradually and was associated with tinnitus and diplopia. An audiometric test indicated a loss of hearing on one side of 69 per cent and on the other of 61 per cent. Thereafter during a period of two months there was great improvement in the hearing with the loss at the end of that time reduced to 21 per cent on one side and 9 per cent on the other.

MacCready[23] in 1937 reported the case of a white boy, age 15 years, to whom a prophylactic dose of 3,000 units of tetanus antitoxin had been administered intramuscularly in the right arm. Nine days later, the patient experienced a violent anaphylaxis characterized by swelling at the site of the inoculation, vertigo, an elevation of temperature and malaise. With the extreme dizziness there was a great tendency to fall. On

the fifth morning after the onset of this reaction the boy awakened with a considerable degree of bilateral deafness. Audiometric examination demonstrated a loss of hearing of 46 per cent in the right ear and 45 per cent in the left ear. There was no improvement thereafter. This author attributed the deafness to an anaphylaxis which he interpreted as due to "a diffuse serous labyrinthitis with a resulting partial destruction of function of both parts of the labyrinth."

A case of permanent nerve deafness owing to an anaphylactic reaction resulting from the administration of tetanus antitoxin recently came under my observation. The report of this case follows.

REPORT OF CASE.

A white man, age 51 years, received a laceration of the forefinger of the right hand on Feb. 20, 1940, while he was working on an air compressor. The wound was sutured, and the patient was immediately given 5,000 units of tetanus antitoxin intramuscularly. He experienced no immediate reaction, but five days later he was awakened from sleep by nausea, headache, general urticaria and pronounced swelling of the lips, ears and scalp. The eyelids were so edematous that he could not see sufficiently well to get about, and he suffered from severe general pain. Within about six hours after these initial symptoms of anaphylaxis, he became aware of an intense ringing in the ears and a rapidly developing deafness. An otologist was immediately called in consultation.

The patient had always been in good health and had at no time experienced trouble with the ears or upper portion of the respiratory tract. He related that his hearing was normal prior to the onset of the deafness following this injection of tetanus antitoxin, and his statement was corroborated by his family and his employers. No history of any allergic manifestation whatsoever nor of hereditary disease or disease of the central nervous system could be elicited. The only relevant information in the history was that seven years previously the patient had received tetanus antitoxin in the treatment of an abrasion.

The general physical examination gave normal results. The Wassermann and Kahn tests repeatedly resulted in negative reactions. The wound healed promptly.

On July 18, five months after the onset of the deafness, the patient first came under my observation. Aural examination revealed a normal external ear; the drum membrane showed the usual light reflex, and there was no greater reaction or displacement of the malleus or umbo than would be expected in a man of the patient's age. He could not hear conversation in ordinary tones at a distance of two feet, but he could hear loud conversation at a distance of four feet.

By air conduction, tuning forks C-64 to C-1,024 were heard with effort in both ears. Air conduction gave better results than bone conduction on both sides. By bone conduction, an A-1 fork was heard for five seconds on each side, as contrasted with the normal period of 20 seconds. The Weber test gave negative results. The greatest loss of hearing was for the high tones, as demonstrated by audiometric examination, and the curve shown in the audiogram was essentially the same for both ears. There was a loss of 68 per cent for the right ear, and for the left ear the loss was 70 per cent.

The Eustachian tubes were patent and could be inflated with ease. Neither nystagmus nor vertigo was present when both ears were irrigated with tap water with the patient upright and then bent forward. Horizontal nystagmus to the left was observed for approximately 35 seconds when the patient in an upright position was turned 10 times to the right and it was noted for about five seconds longer in the opposite direction when the corresponding turning test to the left was made. These tests caused the patient to complain of slight vertigo, but no nausea.

Six times during the next nine months the patient was carefully examined. As each succeeding examination corroborated the observations of the preceding ones, hysterical deafness was ruled out on the basis of the consistent audiograms and the results of the repeated tuning fork tests. In addition, the vestibular tests indicated definite vestibular changes of a degenerative type. The repeated examinations established the diagnosis of nerve deafness that appeared to be permanent.

SUMMARY AND CONCLUSIONS.

In the case presented, a man in good health was given a prophylactic dose of tetanus antitoxin within two hours after

receiving a laceration of the finger. The wound healed normally and was unrelated to later developments. A general anaphylactic reaction developed five days afterward and was followed in approximately six hours by bilateral deafness of a high degree, associated with vestibular changes of a degenerative type. This aural condition has continued without improvement. The permanent perception deafness appears to be without doubt the direct result of the injection of tetanus antitoxin.

The subject of deafness from an anaphylactic reaction has not been studied sufficiently at this time to insure knowledge of the pathological changes which take place. Whether it is caused by a diffuse serous labyrinthitis with partial destruction of the function of both parts of the labyrinth has not yet been definitely determined. Another explanation that may be considered is the compressive neuritis somewhat similar to that of Bell's facial paralysis. It seems possible that an edematous process may occur in some part of the bony groove or perineural sheath of the nerves or their roots that interferes with the blood supply and causes impairment of nutrition or anoxemia with temporary inactivity of nerve cells and parenchyma. In my case there arises the question of previous sensitization, for seven years previously the patient had been given tetanus antitoxin as a prophylactic measure. J. L. McCarthy, Associate Director of the Medical Division of Sharpe and Dohme, insisted in a personal conversation with me that in the treatment of a patient who has previously received horse serum, if an antitoxin serum is indicated, bovine serum should be substituted for horse serum even though many years have elapsed since the horse serum was administered.

It is likely that many otologic complications of serum sickness go unrecognized and that otologic damage occurs more frequently than is realized. It is possible that some patients suffering from various types of meningitis, who suddenly become deaf, may have received injury to the internal ear or cortical centers of the brain from the enormous doses of the serum given in the treatment of these infections rather than from the pathological changes owing to the meningitis. Like injury may occur in cases of deafness associated with diphtheria or scarlet fever when the drum membrane and middle

ear are intact. The otologist may well keep in mind the otological implications of serum sickness.

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PRACTICAL AIDS IN THE TECHNIQUE OF CATHETERIZING, LAVAGING AND INJECTING THE SPHENOID SINUS.*

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Many refinements in the clinical methods of investigating the sphenoid sinus have been developed since Lieutaud first found the evidence of disease in this region as a result of his examination of postmortem material.[1] The use of the nasopharyngoscope, X-ray and lavage of the sinus contents are notable examples. The latter method of investigation is an extremely valuable one and may be undertaken by catheterization of the natural ostium or by direct puncture of the anterior sphenoidal wall according to the method popularized by Watson-Williams.[2] Unquestionably, each of these respective methods has its own relative merits. In our hands, however, the utilization of the natural ostium as a means of gaining access to the sphenoidal sinus has proved highly satisfactory in the majority of instances.[3,4] There are some fine points involved in the details of this technique, the observance of which will often determine the success or failure of this procedure.

THE TECHNIQUE OF PROBING, CATHETERIZING, LAVAGING AND INJECTING THE SPHENOID SINUS.

a. Probing: As a preliminary measure the nose is sprayed with a suitable shrinking agent such as a weak solution of ephedrine sulphate or neosynephedrine hydrochloride, and a short time is allowed to elapse for adequate contraction of the nasal tissues. This is followed by a light spray of a 2 per cent solution of pontocaine hydrochloride. The medial aspect of the middle turbinate and adjacent portion of the nasal septum are best anesthetized with an applicator which has been soaked in the pontocaine solution. After this, a few

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drops of 1-1000 adrenalin applied topically to this area will induce maximum constriction of the tissues and thus insure the greatest possible working space between middle turbinate and septum. The region of the sphenoidal ostium is next anesthetized thoroughly by allowing an applicator to remain in contact with this area for several minutes. Some patients will complain of headache when the applicator first contacts this region, but this discomfort rapidly disappears as the anesthesia develops.

Once the anesthetization is complete, probing for the sphenoidal ostium may be undertaken. For this purpose a Ludwig fine applicator with a slight lateral curve in its anterior third is tipped with a small piece of cotton, dipped in the anesthetic solution and then introduced along Zuckerkandl's line to the anterior wall of the sphenoid. By gentle palpation the ostium is located, and the applicator is inserted through the ostium to the posterior wall, where it is allowed to remain in contact with the mucosa for several minutes. In palpating for the ostium one gains the impression of a shallow depression immediately surrounding the orifice, and when the applicator contacts the posterior sphenoidal wall, there is a distinct sense of solidness imparted to the applicator that cannot be mistaken. When the applicator is withdrawn, the distance between the columella and posterior sphenoidal wall is measured and recorded. This average distance is 9.29 cm. for the right and left sides in the adult male, and 8.92 cm. for the right and 8.94 cm. for the left in the adult female. In both sexes, however, this distance varies between 7 cm. and 11.5 cm., depending on the anteroposterior extent of the sinus and how close to the posterior wall the applicator can be placed.

The method of following along Zuckerkandl's line is safer and less difficult than the method proposed by Sieur and Jacob, in which a Eustachian catheter is passed along the undersurface of the cribriform plate to the anterior sphenoidal wall.[5]. With the former method a knowledge of the distance from the columella to the anterior wall of the sphenoid is not essential, the accepted textbook teaching to the contrary, although it may be of more value if the latter method is employed. The use of the anterior nasal spine as a landmark as advocated by the textbooks is not practical

in the living patient. The columella is far more accessible and more valuable in this respect. Some authors have minimized the possibility of injury to the cribriform plate during the process of probing the sphenoid owing to the hard, compact nature of its bone. It should be remembered, however, that large dehiscences or actual absence of the cribriform plate are not too uncommon.[6]

b. Catheterization and Lavage: After the patient has been completely anesthetized in the above manner, he is placed in the recumbent position, and a five inch silver cannula, which has been shaped to conform with the curve of the applicator, is introduced through the ostium into the sinus. The head of the patient is now extended so as to place the cannula in a vertical plane. The cannula is then grasped near the distal end by the thumb and first two fingers of the left hand with the hypothenar eminence resting on the dorsum of the patient's nose. The cannula is held firmly while the syringe is being attached, in order to prevent rotation and consequent injury to the sphenoidal mucosa. After attachment of the syringe, the cannula is then grasped near the apex of the nose and withdrawn about one-eighth inch from the posterior wall of the sinus. This allows the solution to flow freely and avoids any trauma to the mucosa.

The lavage solution is injected slowly until the moment at which it overflows the sinus and drains into the nasopharynx. The patient should be advised of this beforehand and that the excess solution may be swallowed. The exact amount of fluid required to fill the sinus is noted and the contents quickly aspirated back into the syringe at the precise moment at which the fluid appears in the patient's nasopharynx. This same amount of fluid should be repeatedly reintroduced into and reaspirated from the sinus. This process is properly referred to as churning, and it is a very important part of the procedure. No sphenoidal sinus lavage should be considered complete until the solution has been churned in the sinus at least 20 times.

c. Injection of Opaque Medium: The anesthetization and position of the patient are the same as for the lavage. The cannula is withdrawn about one-eighth inch from the posterior wall of the sinus and held firmly in this position at the apex of the nose while the opaque substance is slowly injected

by means of a 5 cc. sana-lok syringe. When the maximum capacity of the sinus is approached, as determined by noting the amount of fluid the sinus held during the previous lavage, the nasopharynx should be periodically inspected for the appearance of the opaque oil by elevating the soft palate. Patients cannot always be depended upon to determine the presence of the opaque medium in this region. As soon as the oil is detected, the injection should be stopped. The patient is then assisted from the table, care being taken to maintain the head in full extension while this is being done, and he is permitted to sit or stand with the head fully extended for about 10 minutes. He should be requested to swallow every two or three minutes during this period. This allows all the excess solution to drain away from the nasopharynx and sphenoethmoidal recesses. This position is maintained while the patient is being placed on the X-ray table, and X-rays should be made without delay in order to avoid leakage from the sinus. Opaque oil used as a contrast medium seems to have a mild therapeutic effect of temporary nature, and therefore it is not lavaged from the sinus after the Roentgenograms are made.

Choice of Solutions: a. Solutions for Lavage: Some care should be exercised in the choice of a lavage solution, since antiseptic solutions or solutions that are strongly acid or alkaline, or hyper- or hypotonic may interfere with normal ciliary activity and induce a reactionary edema in the mucosa which will more than offset their therapeutic value.[7] If it is desired to make a bacteriologic study from the lavage specimen, sterile normal saline solution or distilled water may be used. Sterile distilled water likewise seems to be the medium of choice for lavage when a cytologic study is needed. For therapeutic purposes in those cases in which the lavage is likely to be repeated frequently, normal saline solution, with or without the addition of small amounts of ephedrine sulfate or neosynephedrine hydrochloride, is the solution of choice. We did not practice irrigating the sphenoidal sinus with antiseptic solutions.

b. Solutions Used as a Contrast Medium: The proper selection of an opaque medium is important and will often determine the success or failure of an injection. Too much stress cannot be placed on this seemingly minor point. If the sphe-

noidal ostium is small or highly situated on the anterior wall, an opaque substance with low viscosity, such as thorotrast, theridol or lipoiodine diagnostic, may be used with success. On the other hand, when the ostium is large or placed lower on the anterior wall than usual, the viscosity of the opaque medium must be relatively high in order to prevent rapid drainage from the sinus into the nasopharynx and sphenoethmoidal recesses before the X-rays can be taken. The presence of an opacity in these regions detracts from the value of the Roentgenogram. In cases of this nature. iodochlorol diluted with an equal amount of pure olive oil, or a suitable mixture of brominol light and brominal heavy, may be used to obviate the possibility of rapid drainage. A sinus smaller than average also requires an oil with a relatively high viscosity.

It is usually preferable to inject one sinus at a time with opaque medium, although in certain cases a bilateral injection may be made at one sitting. In some instances there exists a marked disparity in size between the sinuses of opposite sides, and the smaller, more rudimentary sinus often has an unusually large ostium. In this case it is best never to attempt to inject both sinuses at once, since the oil from the smaller one almost invariably drains into the nasopharynx and thus impairs the diagnostic value of the X-rays. If it is desired, the antrum of one side may be injected at the same time of the sphenoidal injection. The sphenoidal sinuses are first injected, and the usual Roentgenograms are made. The patient's head is kept in full extension while he is assisted from the table and placed in a chair for the antral injection. After the antrum has been filled with opaque substance by inferior meatal puncture, the usual Roentgenograms necessary to outline this sinus are made in the customary manner.

It is essential to vary the viscosity of the opaque substance in accordance with the size and position of the ostium and the topography of the sinus if a high percentage of successful injections is to be maintained. It should be remembered, however, that unless otherwise indicated, a solution of marked density is neither essential nor desirable.

Difficulties Encountered in the Procedure: These are chiefly on a pathological or anatomical basis. Adhesions in the nose from previous operative trauma or disease may often be

overcome by shaping the applicator or cannula in such a manner as to pass through or around them. In some instances they must be removed in the usual manner. An enlargement of the middle turbinate from marked hypertrophy, hyperplasia, polyposis or, more rarely, pneumatization may reduce the available working space between the turbinate body and septum. In many cases a simple hypertrophy of the turbinal mucosa may be overcome by thorough shrinkage. However, when hyperplasia is marked and the response to vasoconstrictors poor, it is necessary to fracture the middle turbinate laterally with a suitable flat-bladed instrument or Killian speculum, or else remove the posterior third. Polyps, when abundant, obscure the customary landmarks and should be removed. A middle turbinate which has been invaded by an aberrant ethmoidal cell must be crushed in order to provide the necessary space within the nasal cavity. Usually these difficulties may be easily overcome.

It is preferable to place the patient in a recumbent position for lavage of the sphenoid sinus, and it is imperative to have this position if an opaque oil is to be injected for diagnostic purpose. This position may add to the difficulty of inserting the cannula through the ostium, however, if one is accustomed to view the nasal cavity with the patient in the upright position. In this case it is advantageous to insert the applicator into the sinus, introduce the cannula into the nose by the side of the applicator, and then withdraw the applicator. With the applicator having served as a landmark, it is then usually an easy matter to insert the cannula into the sinus.

Variations in the topography of the nose will sometimes increase the difficulty or obviate the possibility of a successful sounding of the sphenoidal cavity. A dislocated columella or collapsed ala nasi may make it difficult to view the interior of the nasal cavity in its proper relationship, although it is doubtful if either of these factors alone will make the probing impossible. A high posterior deviation of the septum or a sharp septal spur that projects into the posterior third of the middle turbinate, or both, are common anatomical aberrations found in this region which may well make it impossible to locate the sphenoidal ostium. A submucous resection of the septum may occasionally be necessary to obviate this difficulty.

In some patients the middle turbinate forms a right angle with the ethmoidal capsule instead of the more common obtuse angle and thus serves to block off the region of the olfactory fissure effectively. Even after thorough shrinkage the intervening space between the middle turbinate and septum may not be of sufficient size to permit the passage of an applicator without considerable trauma to the tissues. Such a turbinate must be displaced laterally or removed in its posterior third.

Variations in the size and position of the sphenoidal ostium and slope of the anterior wall of the sphenoidal sinus may facilitate sounding of the cavity or else increase the difficulty of this procedure. The small, highly situated ostium is usually more difficult to reach than the large, more lowly situated one. Dixon recently stressed variations in the ethmoseptal angle, slope of the anterior sphenoidal wall, and drainage point of the sinus as factors which increase the difficulty of catheterization.[8] An ostium which drains into a posterior ethmoidal cell or into the posterior tip of the middle turbinate as in one of Dixon's unique specimens is totally inaccessible for sounding. The same may be true of an ostium placed more laterally than usual or one that is unusually close to the cribriform plate. The ostium of one side is frequently situated at a different level than the ostium of the opposite side, so that while one may be readily accessible, the other may be beyond access. It should also be remembered that the sphenoidal ostium may occasionally be partially or completely closed or absent.[9,10]

In those cases in which the ostium cannot be located and in which there is sufficient working space between the middle turbinate and septum, the method of Tremble may be used to advantage.[11,12] This consists of puncturing the anterior sphenoidal wall with his newly devised sphenoid cannula. We used this method in a number of instances and believe it to be safe if properly employed. Before attempting the procedure, however, it is well to secure a preliminary Roentgenogram of the sphenoid sinus in the Hirtz and lateral positions in order to obtain some idea as to the general topography of this region. It is perhaps best that this method supplement but not supplant the usual method of catheterizing the sphenoid sinus with a cannula.

Occasionally, the anterior sphenoidal wall may be so thick

as a result of irregular pneumatization or a sclerosing type of bone pathology that one must resort to a drill in order to gain entrance into the sinus. Smith[13] recently described a modified Thornwaldt drill to be used for this purpose, and while we have not yet used the instrument in our work, it seems likely that it may be of value in this type of case. Here again, one should be thoroughly familiar with the topography of the region before attempting to puncture the anterior sphenoidal wall by this method.

The mucosa on the posterior wall of the sphenoidal sinus should be thoroughly anesthetized by allowing the applicator which has been soaked in the anesthetic solution to remain in place here for several minutes as previously mentioned. If this is not done, the lavage solution or opaque oil, since it impinges with greatest force on that area just beneath the tip of the cannula, will often induce a sensation of pressure in the head which some patients will not tolerate. This difficulty can almost always be overcome by adequate anesthetization.

When there is marked obstruction in the nasal passages with a laterally situated ostium, it may be necessary to impart such a lateral curve to the sphenoid cannula that the tip just protrudes through the ostium and thus contacts the lateral rather than the posterior wall. In this case the sinus is easily filled with the lavage solution, but only a small part of the solution can be aspirated due to the fact that the cannula tip can be introduced only a short distance through the orifice. It is then necessary to correct the nasal deformity responsible for this situation or else puncture the anterior wall if a successful lavage of the sinus is to be made.

Pus in the sphenoidal sinus often collects into relatively firm gelatinous masses which tend to resist disintegration. This is especially likely to occur when there are pockets formed by accessory septa or extensive pneumatization into the surrounding structures. Under these conditions there must be repeated churning of the lavage solution in the sinus cavity in order to break up these semisolid masses so that they may be aspirated back into the syringe. Until this happens, the lavage returns may be perfectly clear and thus give the impression of a negative lavage. In numerous instances we have seen a sinus, at first thought to be normal because

of the first clear lavage returns, converted into a pathological one yielding an abundance of pus when the proper churning technique had been carried out. The process of prolonged churning is an extremely important one and, therefore, cannot be overemphasized.

The injection of the opaque substance may cause some difficulty if the cannula is too long or the syringe too large. A five inch silver cannula and a 5 cc. sana-lok syringe are ideal for this purpose. If an opaque oil with high viscosity is indicated, it may be necessary to exert considerable force on the plunger of the syringe in order to expel the contents. This not only requires a great expenditure of physical effort but also increases the danger of the procedure. This difficulty may be overcome to a great extent by always selecting an oil with the lowest possible viscosity that is consistent with a successful injection and by placing the oil in hot water about 10 minutes immediately prior to the injection. The warm oil is expelled easily from the syringe if the injection is made rapidly.

Complications of the Procedure: The possibility of complications from this procedure may be reduced by careful technique. While there is undoubtedly mild trauma to the middle turbinate and adjacent septum and the region of the sphenoidal ostium in most cases, we did not encounter any case in which there was evidence of undue trauma in these areas. Occasionally, the tissues immediately surrounding the sphenoidal ostium may be lacerated with the formation of a drop or two of blood which enters the sinus cavity and serves to discolor the subsequent lavage.

The possibility of pushing an applicator or cannula through a thin, diseased or dehiscent sphenoidal wall may be minimized by gentleness in palpation and by holding the cannula firmly and out of contact with the sinus wall while making the lavage or injection. In one instance brought to our attention the cannula had apparently been inserted through the roof of the sphenoid, and about 1 cc. of opaque oil was injected into the middle cranial fossa. There were no undue effects from this except headache which lasted for about four or five days.

The periodic discharge of oil from the sinus for one or two

days subsequent to the injection may cause nausea in some individuals, not as much from any inherent property of the oil as from the unpleasant association with castor oil. Ordinarily, the sphenoid sinus should empty itself in about 72 hours. One patient in our series retained oil in a pocket formed by an accessory septum for an entire year, while another retained opaque medium in a pneumatized pterygoid process for eight months.

Some patients complain of headache during lavage or injection. This is more likely to occur if the anesthetization of the sphenoidal mucosa is incomplete or if the injected solution is of high viscosity. Usually, however, the headache is short-lived and frequently simulates the headache which the patient has been accustomed to having. Proetz emphasizes the fact that patients who complain of severe headache with the first few treatments by the displacement method usually obtain the best therapeutic result. This was certainly true in several patients in our series.

It rarely happens that a large posterior ethmoidal cell may be lavaged instead of the true sphenoidal sinus. Unless the lavage is followed at a subsequent date by an injection of contrast medium, and X-ray, it is impossible to tell when this has been done, since the opaque substance is usually injected into the same cavity that is lavaged. This error will, of course, spoil the Roentgenograms and necessitate a repetition of the procedure but does not cause the patient any undue effects.

SUMMARY.

In this paper we have attempted to point out some of the more significant practical aids which were of benefit to us in developing a successful technique for probing, lavaging and injecting the sphenoid sinus. Emphasis was placed on gentle technique, proper anesthetization, recumbent position of the patient and frequent churning of the lavage solution in the sinus. Comment was made on the proper choice of a solution for lavaging the sinus, and important factors concerned in the proper selection and injection of an opaque medium were discussed. Some of the more common difficulties encountered were noted, as well as some of the poten-

tial complications of the procedure. The technique we have described and are now employing was evolved in an effort to insure the patient a minimum degree of discomfort and a maximum degree of safety from the procedure, as well as to provide valuable clinical information.

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CRICOPHARYNGEAL SPASM.*

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The term cricopharyngeal spasm often is used synonymously with functional dysphagia, spasmodic stenosis, globus hystericus, hysterical dysphagia and esophageal neurosis. It commonly is described as a "lump in the throat" and as a symptom of disease of the upper esophagus. The difficulty in differentiating between functional disturbances and organic diseases at the level of the cricopharyngeus muscle probably has contributed to this apparent state of confusion. In addition, the lack of interest manifested by physicians in diseases of the upper end of the esophagus and their tendency to ascribe symptoms of an indefinite character to functional or emotional disturbances has been an added factor. It would seemed desirable, therefore, to review this subject and study the behavior of the cricopharyngeus.

The cricopharyngeus muscle consists of the lowermost or transverse fibres of the inferior constrictor muscle of the pharynx. It is attached laterally to the cricoid cartilage and is inserted posteriorly in the median raphé of the pharynx. It can be considered as a sling-like muscle which surrounds the posterior and lateral aspects of the cricoid cartilage and the upper orifice of the esophagus. It *does not encircle* the esophagus. Above, it is continuous with the oblique fibres of the inferior constrictor of which it is a part; below, it merges with the circular muscle fibres of the esophagus. The circular muscle consists of striated or voluntary fibres and has an innervation similar to that of the cricopharyngeus. The inferior constrictor muscle including its transverse fibres as well as the circular muscle of the upper esophagus are innervated through the pharyngeal plexus which receives nerve fibres from the glossopharyngeal, vagus and sympathetic nerves.

While the cricopharyngeus, which commonly is considered as the sphincter of the upper end of the esophagus, plays an

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important part in the act of swallowing, it apparently makes no contribution towards propelling food from the pharynx into the stomach. This function is accomplished by the action of the three constrictor muscles of the pharynx, as well as the muscles of the esophagus. At the approach of a bolus of food, the cricopharyngeus relaxes and allows it to enter the esophagus. According to Negus, an important function of this muscle is to keep the esophagus tonically closed to prevent air from being inspired into the esophagus, where it would serve no useful purpose and, further, would interfere with normal respiration, in that it would cut down the tidal volume of respired air. It is very probable that the circular muscles of the esophagus subjacent to the cricopharyngeus also serve a similar function, for in laryngectomized patients in whom the cricopharyngeus has been severed from its attachments to the cricoid cartilage there normally is little aerophagia or distention of the esophagus (see Figs. 1 and 2).

While the action of the cricopharyngeus can be studied by direct endoscopic and Roentgen ray examinations, slight disturbances usually cannot be observed as the process of swallowing is rapid in this location. Marked narrowing of the lumen, the result of physical changes, can be readily detected in the lateral and in the posteroanterior position, as the stream of barium will be seen to be narrowed. To ascertain the presence of slight changes, Mosher has emphasized the importance of centering the fluoroscopic screen or the film on the larynx.

While esophagoscopic examination commonly is employed to ascertain the presence of disease or spasm of the esophagus, a considerable experience is required to distinguish between the normal and the presence of spasm in an unanesthetized patient. Usually the sphincter remains tightly contracted and will relax only on swallowing or on deep inspiration. General anesthesia or profound narcosis will relax the normal cricopharyngeus and thus assist in differentiating between the normal and the spasmodically contracted or organically narrowed cricopharyngeus.

Although Elze believes that the true functional constriction producing the characteristic stellate lumen as seen esophagoscopically is formed by the upper circular fibres of the esophagus, a majority of esophagoscopists consider the upper sphinc-

Fig. 1. Roentgenograms made in case of man who had laryngectomy for carcinoma seven years previously. In the postoperative repair of the pharyngeal stoma an attempt was made to approximate the cut ends of the inferior constrictor muscle, including the cricopharyngeal portion. The

ter of the esophagus as the cricopharyngeal fold which is produced by the cricopharyngeus and is posterior to the lumen of the esophageal orifice. It is for this reason that insistence is made on keeping the lip of the esophagoscope anteriorly when passing through the cricopharyngeal constriction. It must be admitted, however, that the upper circu-

Fig. 2. Lateral Roentgenogram reveals constricted portion of food passageway below seventh cervical vertebra. This exhibited no organic narrowing and readily admitted a 10 mm. full lumen esophagoscope. Its location and behavior corroborate the opinion that the circular muscle fibres of the upper esophagus act as a sphincter or constrictor. (Film by Dr. S. L. Caspar.)

lar fibres *do exert* a constricting effect since the lumen of the cervical esophagus does not balloon up as readily as the thoracic esophagus when the cricopharyngael fold is passed.

Since the normal function of the cricopharyngeus is to maintain closure of the upper end of the esophagus and to relax only to permit passage of fluids and food, any abnormal contraction or spasm will interfere with its relaxation during

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patient has developed an excellent pseudovoice. There is no dysphagia. To rule out a possible stenosis of the hypopharynx or esophagus, a 10 mm. full lumen esophagoscope was passed. (A) reveals a large air-filled space extending to the second thoracic vertebrae (T-2).

(B) the food passageway is narrowed at the upper border of the first thoracic vertebrae suggesting the presence of a sphincter-like muscle. The barium column (C) is completely interrupted at this point, indicating that there is present some form of sphincter or constrictor. Its location at the level of the upper border of the first thoracic vertebra (T-1) indicates that it is not the cricopharyngeus. (Films by Dr. S. L. Caspar.)

the act of swallowing, or may give rise to painful sensations referred to the region of the upper end of the esophagus. It is apparent that any act as complex as swallowing would be influenced by a wide variety of stimuli to any one of the central or peripheral nerve pathways. Since the circular muscle of the upper portion of the esophagus is made up of striated fibres which are closely related to the cricopharyngeus and have practically a common nerve supply, it should be necessary to consider this in discussing spasm. It also would appear necessary to ascertain whether there is excessive muscular contraction present constituting true spasm or whether the disturbances are due to failure to open a tonically closed normal sphincter. There is no known method of examination which will afford this information. Beatty and Palmer have referred to certain patients who cannot swallow tablets or capsules and ascribed their difficulty to faulty management of the tongue or oral muscles in the act of deglutition. As soon as the object reaches the pharynx it is swallowed without difficulty. Small quantities of barium often are retained in the hypopharynx for brief periods during a functional study of the esophagus. This cannot be interpreted as spasm since a considerable portion of the mixture already has passed into the esophagus. Temporary lodgment of a barium-filled capsule in the esophagus often is constructed as spasm. This is especially common in the Roentgen ray study made following removal of an esophageal foreign body. There usually is present inflammation and edema which can interfere with the passage of a capsule and also may initiate spasmodic contraction of the circular muscle fibres.

It is difficult to accept cricopharyngeal spasm as the cause of lodgment of foreign bodies in the cervical esophagus. A majority of these objects are below the cricopharyngeus and are held by the circular muscle fibres of the esophagus or more probably by a combination of muscular contraction and a crowding of the esophagus at the upper narrow aperture of the thorax by periesophageal viscera. It is a common experience to have the foreign body descend rapidly when once dislodged from the cervical esophagus.

Nonstriated muscle fibres which form the walls of practically all of the contractile tubings and sacs of the body, and a majority of the sphincters which guard these are frequently

the seat of spasm. While this form of misbehavior is not so common in striated voluntary muscle fibres, it can and does occur, as is evidenced by the larynx when foreign material is aspirated into the airway. Nonstriated muscle fibres are definitely susceptible to psychic factors. Faulkner and his associates have demonstrated by Roentgen ray examination, as well as by esophagoscopy, that disturbing emotional influences appear to be underlying factors in the production of esophageal relaxation or spasm. They were able at will to produce esophageal spasm by inducing thoughts of insecurity and frustration, while suggestions of a pleasant character caused esophageal relaxation.

In a review of the literature on disturbances of the cricopharyngeus muscle, one is impressed with the many conflicting reports. In the syndrome designated as Plummer-Vinson's, the cricopharyngeal narrowing has been described variously as being due to functional stenosis, webs or changes in the mucosa and submucosal layers. Vinson considered th disturbances as functional. Mosher stated that in a majority of the patients he examined there was a physical basis for the symptoms, namely, a web, and that treatment of this gave prompt relief. While Kelly and Paterson believed that the disturbances may be due to spasm, Paterson observed that a loss of the normal sphincter-like form of the mouth of the esophagus is not unusual, the opening appearing as a pin hole, a slit or an irregular opening. There is a feeling of resistance when the esophagoscope is passed, which probably is due to a lack of resiliency, the result of thickening of the submucosa, as was observed by him in the lingual and buccal mucosa. Rodgers believed that the innervation of the sphincter is under control of the sympathetic component of the pharyngeal plexus, derived entirely from the superior cervical ganglion. He successfully treated such a case by sympathetic denervation and reported an excellent functional result. We are of the opinion that there is definite organic disease at the cricopharyngeal level in these cases and while there may be an element of spasm present, this does not account for the dysphagia nor can relief by esophagoscopy be explained on this basis alone. This difference of opinion by a number of observers suggests that visual examination of the upper orifice of the esophagus is not entirely satisfactory and that it would be extremely difficult to detect by this means alone failure of a normal appearing cricopharyngeus to relax.

One often is tempted to make an inferential diagnosis in patients who complain of indefinite, indescribable sensations about the throat which may be associated with the swallowing of food and often are more distressing when saliva alone is swallowed. Many of these patients already have been diagnosed as cases of hysteria, and the question immediately arises whether it would be desirable to carry out further investigations, including esophagoscopy, and by so doing "fix the neurosis" or to ascertain if the functional disorder is due to organic disease. The following case is cited:

Case 1: A woman, age 48 years, employed for many years as a school teacher, complained of a lump in her throat which could not be localized accurately, did not interfere with swallowing of food, was equally distressing when saliva was swallowed and was aggravated by emotional upsets and physical fatigue. She exhibited evidences of a neurotic personality and presented certain of the so-called "stigmata" of hysteria. This diagnosis appeared to be amply justified. A Roentgen study of the swallowing function was not satisfactory, due to her failure to co-operate. The desirability of esophagoscopy was debated but finally was performed and an early postcricoidal carcinoma was found.

Comment: The growth was not obstructing sufficiently to interfere with the passage of food. Its presence apparently excited reflex contractions of the striated circular muscle fibres of the cervical esophagus which are supplied by the same nervous mechanism as is the cricopharyngeus and these accounted for the subjective disturbances in the swallowing function.

It is probable that reflex effects may be referred from more distant points. Instances of symptoms referable to the upper esophagus in diseases of the stomach, duodenum or biliary apparatus have been cited. The following case is illustrative:

Case 2: A man, age 54 years, who recently had sustained serious financial losses, complained of attacks of difficulty in swallowing, which he referred to the upper esophagus. These attacks came on without warning, were of brief duration, unassociated with pain and suggested complete obstruction of the esophagus since the patient was unable to swallow even his saliva during these episodes. A Roentgen ray study

of the swallowing function revealed a normal cervical and upper thoracic esophagus. There was observed some change in the lower third of the esophagus which suggested ulceration, as a small quantity of barium adhered to the esophageal wall. The patient failed to respond to antispasmodics and general medical care and finally esophagoscopy was recommended. Examination of the esophagus revealed an early carcinoma involving the anterior wall at the junction of the middle and lower thirds. This was nonobstructing and apparently did not interfere with the passage of food.

Comment: It was believed that irritation of the carcinoma was responsible for the reflex disturbance in the upper esophagus, which was interpreted as failure of the cricopharyngeus to relax during the swallowing act, with possibly contraction of the circular esophageal fibres. Whether it was due to increased contraction of these muscles or to a dissociation of the swallowing mechanism could not be determined. There was nothing to suggest a diagnosis of hysteria.

A similar mechanism has been suggested as responsible for the development of pulsion diverticulum of the pharynx. Inco-ordination of the swallowing mechanism, either by failure of the cricopharyngeus to relax or increased tonic contraction of its fibres, would temporarily prevent the progress of a bolus of food which is being propelled onward by the forceful contractions of the pharyngeal constrictors. This could be compensated for only by a bulging or stretching of the pharyngeal wall, particularly at the weak point in the inferior constrictor muscle between its circular and oblique fibres posteriorly. Patients occasionally describe a momentary "cramp" of the throat muscles, especially when swallowing fluids, and often have a severe choking attack due to aspiration of fluids. It is conceivable that this would occur in cases of chronic hypopharyngitis.

We have observed several cases of disturbed swallowing unassociated with obstruction which could not be explained by Roentgen ray examination and esophagoscopy. All were middle-aged men who exhibited certain neurotic manifestations and could be diagnosed as functional dysphagia. Systemic studies revealed evidences of moderate hypothyroidism. The administration of thyroid extract promptly relieved all symptoms.

COMMENT.

With our present diagnostic aids it is difficult to demonstrate dysfunction of the cricopharyngeus muscle. In the presence of disease of the cervical esophagus, as carcinoma or foreign body, and in chronic hypopharyngitis it is reasonable to assume that inco-ordination of the swallowing function does occur; however, it is not always possible to differentiate between this and the changes produced by the disease itself. Further, it cannot be ascertained whether reflex stimuli are producing increased involuntary tonic contractions (spasm)) of the cricopharyngeus or there is lacking the necessary stimulus to relax the normally contracted sphincter. In addition the circular muscle fibres of the upper esophagus, which are striated and receive practically the same innervation as the cricopharyngeus, have a related function and probably participate in the mechanism of inco-ordination of the upper esophageal constrictor. One, therefore, should consider in the differential diagnosis, carcinoma, foreign body, pulsion diverticulum, congenital or acquired stenoses, chronic hypopharyngitis, chronic esophagitis, cardiospasm, disturbances of the thyroid gland and a host of other conditions about the neck and upper mediastinum. Certain laryngeal disturbances also may give rise to indefinite symptoms referable to the swallowing act.

CONCLUSIONS.

Cricopharyngeal spasm, also referred to by many other designations, frequently is employed to describe a large group of disorders of the upper orifice of the esophagus.

Dysphagia is one of the manifestations of tension which may be present in any of the neuroses. It also is a common symptom of organic disease of the hypopharynx and esophagus and may be observed in certain disorders of the larynx and periesophageal structures.

A diagnosis of cricopharyngeal spasm or other functional disorder should be made only after other diagnostic possibilities have been excluded by a thorough study.

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CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION.

A sum of money is now available from the Casselberry Fund of the American Laryngological Association for a prize award, decoration or grant to be given for original investigation in the art and science of Laryngology or Rhinology. Theses must be in the hands of the Secretary, Dr. Arthur W. Proetz, 1010 Beaumont Medical Building, St. Louis, Mo., before March 1, 1943.

PATHOLOGIC CHANGES OF THE TEMPORAL BONE IN OSTEOMYELITIS OF THE SKULL.*

DR. HANS BRUNNER, Chicago.

In 1907, Siebenmann[1] presented a classical review concerning the deafness associated with osteomyelitis. There were seven cases at his disposal, three of them being his own. He found that either in the first weeks of an osteomyelitis or from one to three and one-half years after the onset of the acute symptoms, tinnitus, dizziness and a diminution of hearing may suddenly occur. These symptoms, as a rule, are bilateral and may lead to complete or incomplete deafness. In the less severe cases the deafness may be transitory or even improve spontaneously. In cases with complete deafness the labyrinth as a rule does not respond to the different stimuli.

In several instances Siebenmann found an otitis media which was characterized by its painless, torpid course and by the finding of staphylococci in the pus.

In recent years, Theissing[2] and Ersner and Winston[3] have described more cases of this type which essentially confirm the findings of Siebenmann.

According to Siebenmann, the pathology of the deafness in osteomyelitis consists of a purulent labyrinthitis rather than of a neuritis of the cochlear nerve.

I have been able to find in the literature only two cases that illustrate the pathology of deafness in osteomyelitis. The first case, described by Steinbruegge (quoted by G. C. Mueller[4]) was of a man 20 years old, who suffered from an osteomyelitis of the humerus and became deaf. Four years after the onset of the deafness the patient died. The temporal bones were very hard and presented a healed purulent labyrinthitis with development of connective tissue and newly formed bone within the scales and the semicircular canals. The nerves were to a great extent replaced by connective tissue. Quite different were the findings in Brock's[5] case of a

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man, 36 years old, who suffered from an osteomyelitis of the femur and who 12 days before death became deaf in both ears, without dizziness and without loss of labyrinthine excitability. Microscopically there was a mild acute otitis media on both sides. In the internal ears the cochlear nerve as well as the spiral nerve presented spindle-like thickenings; there was, however, no other *definite* pathology in the internal ear, inasmuch as it could not be stated whether or not the changes of Corti's organ actually took place during the life of the patient. Brock considers these changes as parenchymatous degeneration of the cochlear nerve, the spiral ganglion and Corti's organ due to the influence of bacterial toxins, and claims that parenchymatous degeneration had produced the deafness of the patient.

I have observed the following case:

L. K., 37 years old, white, male. After a radical operation of the right maxillary and ethmoid sinus the patient developed an acute osteomyelitis of the skull which required the resection of the entire frontal squama and of the greater part of the right half of the skull (see Fig. 1). About four months prior to his death he complained of pains in the right ear when masticating. There were granulations and pus in the right external canal so that the drum could not be seen. The diagnosis of an osteomyelitis of the bony external canal was made, since the hearing was not markedly impaired and there was no tenderness around the external canal such as characterizes a common external otitis. However, five days later, on July 30, 1940, there was a marked edema over the right temporal squama and there was a typical mastoiditis on the right side. The right external canal was filled with granulations and there was a definite impairment of hearing on the right side.

On Aug. 8 a radical mastoid operation was performed. A large subperiosteal abscess was found over the right mastoid and there were two osteomyelitic areas, one in the middle of the mastoid planum and another in the area of the superior angle of the pyramid. The mastoid process contained many granulations, but little frank pus. However, there was much pus in the antrum containing pneumococci (type I), and there was a sequestrum of the base of the zygomatic bone, the tegmen and part of the floor of the middle fossa. The dura of the middle fossa was covered with granulations. After having finished the radical operation, a large skin flap was reflected downward. A cross incision was made in the temporal muscle, the latter being partly necrotic, due to a purulent myositis. There was an osteomyelitis of a large portion of the temporal squama and this was removed. In the pus, Gram positive diphtheroids were found. On Nov. 10 there was a complete deafness on the left side. The left drum was normal. On Nov. 18 the patient expired.

At autopsy, a large defect of the skull was found, corresponding to the previous surgical procedures. Over the right mastoid area there was a partly healed scar, in the center of which was a fistulous opening, filled with purulent material. This led into the right middle ear and mastoid region. The dura was firmly adherent to the bony structures and over the base of the cranial cavity was spread a thick film of purulent exudate which extended inferiorly to surround the cervical cord. In the right cerebellopontile angle the cerebellar hemisphere was firmly adherent to the posterior surface of the petrous pyramid by thick, fibrinous

exudate due to a purulent pachymeningitis interna. The interpeduncular fossa of the brain was filled with purulent material and the leptomeninges were greatly thickened.

MACROSCOPIC EXAMINATION OF THE TEMPORAL BONES.

Right Side: There was an intense pachymeningitis interna of the posterior surface of the pyramid, involving the internal meatus and ensheathing the nerves. The internal meatus

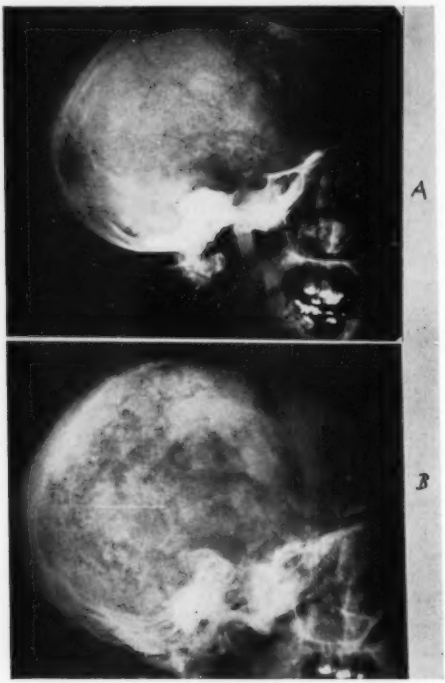

Fig. 1. X-ray film (A) taken on Aug. 3, 1940; X-ray film (B) taken on Oct. 18, 1940. Note the progress of osteomyelitis.

was filled with purulent material, the tip of the petrous bone was diploetic, but apparently normal. The antrum was filled with granulations and purulent material. The sigmoid sinus showed a marked purulent thrombophlebitis.

Left Side: There was a mural thrombosis in the sigmoid sinus. The internal meatus and its nerves were normal and

so was the superior petrosal sinus and the anterior surface of the pyramid. The tip of the petrous bone was diploetic, but apparently normal. The carotid artery and the jugular bulb did not evidence pathology. The middle ear and to a slight extent the mastoid were filled with purulent material, but the tympanic membrane was intact.

MICROSCOPIC EXAMINATION OF THE TEMPORAL BONE.

Right Side: Middle Ear: The mucous membrane is much thickened and forms polypoid excrescences. The thickening is due to an intense infiltration, particularly marked in the region of the windows, and to cysts which contain a serous exudate, frequently opening toward the surface of the mucous membrane. The epithelium is of a columnar type. The mucous membrane of the antrum reveals the same changes.

Of the ossicles, only the short process of the incus can be seen, which does not show any pathology.

The inferior part of the drum is present and evidences a marked thickening due to granulation tissue and the connective tissue layer, the latter forming a clue as to the presence of the tympanic membrane, The drum is covered by squamous epithelium on its outside and by cubical epithelium on its inside, both type of epithelium adjoining one another, at the margin of the perforation of the drum.

The membrane of the round window is perforated by the suppuration in its entire insertion. There is also a perforation of the annular ligament and the footplate of the stapes.

Membranous Inner Ear: The entire membranous inner ear is destroyed by the suppurative process, some shreds of membranes being embedded in the pus. The aqueductus cochleae is free of pathology, but all channels of the modiolus are filled with pus, the latter eroding parts of the bony modiolus.

There are two encapsulated abscesses in the internal meatus (see Fig. 2), one between the cochlear and facial nerve and another between the facial nerve and the roof of the internal meatus. There is no pus within the facial canal, although the osteomyelitic process reaches the periosteum of the descending portion of the facial canal. There is no infiltration of the facial nerve; there is, however, a marked infiltration

of the cochlear nerve and of all branches of the vestibular nerve, extending to the endings of these nerves. The ductus endolymphaticus is filled with pus and the remnants of the endolymphatic sac are embedded in a large abscess on the posterior surface of the pyramid (see Fig. 3).

Bony Capsule of Inner Ear: There is an extensive osteomyelitis which involves particularly the periosteal capsule, while the enchondral capsule is only slightly eroded and the endosteal capsule is almost intact. Only in the region of the semicircular canals, where there is no endosteal capsule, the granulation tissue reaches the endosteum. That actually the

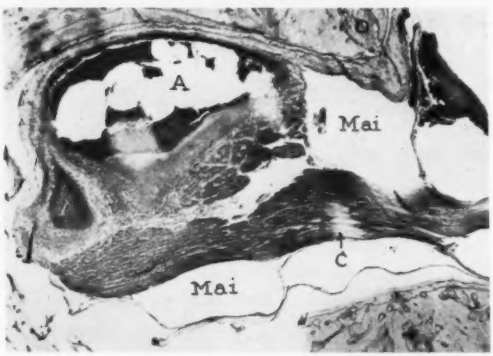

Fig. 2. Section through the internal auditory meatus (Mai) on the right side. (o) Osteomyelitic area in the roof of the meatus, (c) cochlear nerve, (A) abscess within the internal meatus.

periosteal capsule almost exclusively is affected by the osteomyelitis can readily be recognized in the area of the cochlea where the three layers of the bony capsule are easily distinguished. In the area of the semicircular canals the structure of the bony capsule is not as clear, since the periosteal bone penetrates deeply into the inside of the pyramid, occupying a part of the space beeween the semicircular canals. Consequently, in the area of the semicircular canals the osteomyelitis does not only involve the surface of the pyramid but penetrates deeply into the inside of the pyramid, in some places extending even to the endosteum of the semicircular canals.

A second pathway which led the infection into the inside of the pyramid are the blood vessels of the fossa subarcuata.

As a matter of fact, the firm connective tissue of the fossa is replaced either by granulation tissue or by pus (see Fig. 3).

The most intense destruction due to osteomyelitis is found in the superficial layers of the pyramid, particularly on its posterior surface facing the posterior fossa (see Fig. 3). In this area there are large spaces in the periosteal capsule filled with granulation tissue and osteoclasts, the latter destroying

Fig. 3. Section through the internal ear on the right side. (A) Mastoid antrum, (vPy) anterior surface of the petrous pyramid showing osteomyelitis. In (a) newly formed bone bulges into the perilymphatic space of the superior semicircular canal (fSc); (FS) fossa subarcuata, (HC) horizontal semicircular canal, (Cc) crus commune, (D) dura of the posterior fossa beneath which there is an osteomyelitis area, (b) abscess of the bone extending toward the dura, (Ab) abscess of the bone surrounding the saccus endolymphaticus (Se); (F) descending portion of the facial nerve.

the surrounding bone. In many places the granulation tissue invades the dura of the posterior fossa, producing a pachymeningitis, while in other places the inflammation penetrates the dura, invading the subarachnoidal spaces and producing multiple fistulas through the dura. A large area of granulation tissue is found in the roof of the internal meatus (see Fig. 2), while in the area of the saccus endolymphaticus there is a collection of pus between dura and bone (see Fig. 3).

Even in the osteomyelitic areas close to the posterior surface of the pyramid there are spaces which are not filled with

granulation tissue, but with connective tissue, not containing osteoclasts which destroy the bone, but osteoblasts which lay down new bone. Such areas of prevalent osteogenesis are found particularly close to the anterior surface of the pyramid facing the middle fossa (see Fig. 3), at the superior angle of the pyramid beneath the superior petrosal sinus and on the inferior surface of the pyramid. In these areas there is frequently a network of newly formed bone, termed osteophytic and significant at least for a transitory cure of the osteomyelitis. Therefore, there are no fistulas of the dura of the middle fossa, none leading into the superior petrosal sinus or toward the base of the skull.

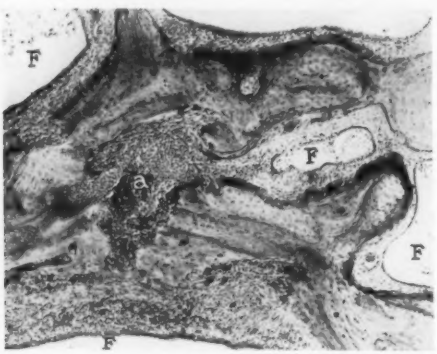

Fig. 4. Section through the mastoid process on the right side. (Z) Pneumatic cells. The mucous membrane is either edematous or changed into granulation tissue. In (a) granulation tissue with osteoclasts invades a bony septum between two pneumatic cells.

Mastoid Process: In the cortical portion of the mastoid process the osteocytes were absent and the channels of the blood vessels were empty to a great extent. This necrosis was due to the fact that the periosteum on the outside was separated by the subperiosteal abscess and the blood vessels originating within the mastoid process were obliterated by the mastoiditis. Thus, the cortex was devoid of blood supply and became necrotic.

The mastoid process contained pneumatic cells, only a few of them being normal (see Fig. 4). In other cells the mucous membrane was edematous, the lumen filled with a serous

exudate, and the bony margins of the cells were smooth. The Haversian channels close to these cells were enlarged and filled with connective tissue and capillaries. The walls of these channels were covered with osteoid substance and there were no signs of resorption of bone. Close to such areas there were other cells, the mucous membrane of which was changed either into granulation tissue or into connective tissue. Occasionally this tissue filled the entire lumen of the cell, changing the pneumatic cell into a "secondary marrow space." The bony walls of these spaces were intensely eroded by osteoclasts, so that in several places the bony septa between two neighboring cells were perforated. The Haversian channels close to these sites were changed into Harversian spaces with intense bone resorption of the walls. It must be emphasized that pneumatic cells with edematous mucous membrane and definite bone deposition on the one hand, and "secondary marrow spaces" with definite bone absorption on the other, were adjacent to one another.

Left Temporal Bone: Middle Ear: Although the greater part of the middle ear was destroyed at autopsy, the following findings could be ascertained: The anterior part of the drum is entirely normal, the posterior part is lacking. In the region of the Eustachian tube the mucous membrane is normal. In the roof of the Eustachian tube, as well as on the anterior surface of the pyramid, there are large marrow spaces filled either by a mixed marrow or by a firm connective tissue. There is no evidence of inflammation.

In the anterior part of the middle ear there are many pneumatic cells in the roof extending to the anterior surface of the pyramid. Several of these cells have an edematous mucous membrane and only in few is there purulent exudate although there is definite inflammation present. The mucous membrane covering the promontorium is normal. The findings are different in the posterior part of the middle ear; *viz.*, in the area of the *aditus ad antrum* and in the antrum. The mucous membrane of the recessus of the windows shows a marked infiltration and in both there are plugs of fibrinous pus. The footplate of the stapes reveals deep Howship's lacunae; however, neither the footplate nor the membrane of the round window are perforated. The inflammation is maximum in

the mucous membrane of the antrum, where in several places even the epithelium is destroyed. In many places osteoclasts are seen destroying the bony walls of the periantral cells, thus carrying the infection into these cells. Only the pneumatic cells close to the mucous membrane of the antrum are infected; the deep perilabyrinthine cells, the pneumatic cells along the posterior surface of the pyramid and the sublabyrinthine cells are normal or only slightly inflamed. On the roof of the jugular bulb there is a small layer of newly formed bone.

Membranous Inner Ear: The membranous inner ear shows a great number of artifacts. Corti's organ is abnormal throughout. It is either absent or it is changed into a layer of indifferent cells. Corti's membrane is either depressed into the internal spiral sulcus or touches Corti's organ. Reisner's membrane is in normal position. The stria vacularis is normal and the scalae do not contain pathological material. There is no exudate in the channels of the modiolus and the capillaries of the modiolus do not present any signs of inflammation. However, there is a definite atrophy of the spiral nerve and the spiral ganglion of the inferior part of the basilar coil. The aqueducts are patent, the maculae and the cristae show changes which are artifacts but show no definite pathology.

Bony Capsule of the Inner Ear: The bony capsule is normal except the internal meatus, where many osteoclasts can be found between the dura and bone. This finding is rather common in cases with meningitis. The nerves in the internal meatus are normal except the cochlear nerve, which shows small, interstitial infiltrations of cells resembling lymphocytes and plasma cells (see Fig. 5). The serial sections prove that these infiltrations originate from the capillaries; *viz.*, that they are perivascular infiltrations. The ganglion of Scarpa and the geniculate ganglion are normal

COMMENT.

The case described here differs from those reported in the literature in having an osteomyelitis of the skull and not of the skeleton; thus the possibility that the temporal bone was infected simply by continuity of the disease must be con-

sidered. This was undoubtedly the case on the right side. On the left side, however, an extension of the disease could not be found microscopically and it must be assumed, therefore, that the infection of the left temporal bone took place by way of the blood stream; that is, in the same way as the infection was reported to have been carried in the cases recorded in the literature. Our case, therefore, offers an opportunity to study osteomyelitis of the temporal bone due to direct extension as well as that due to a blood stream infection.

When the temporal bone becomes affected by direct extension, the infection first spreads from the temporal squama into the bony external canal, the latter being a part of the temporal squama. Osteomyelitis of the bony external canal

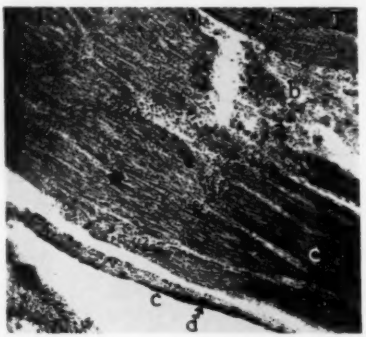

Fig 5. Section through the internal meatus on the left side. (F) Facial nerve, (C) cochlear nerve within the internal auditory canal, (b) capillary with perivascular infiltration, (c) arachnoidea with corpora arenacea (d), (a) infiltration invading the cochlear nerve.

presents similar features to those occasionally observed in epitheliomas of the external canal. From the external canal the disease spreads into the middle ear producing an otitis media and into the mastoid process giving rise to a mastoiditis. As mentioned by Siebenmann, the entire course of the disease is slow and painless, but it can also be rapidly progressing and devastating, resembling infections of the middle ear in the presence of systemic diseases such as diabetes, scarlet fever, etc. The profound deafness on the right side was conspicuous in our case. This undoubtedly was due to the purulent diffuse labyrinthitis. The labyrinthitis was due to the

perforations of the windows but it was not produced by the osteomyelitis of the petrous pyramid, since the osteomyelitis of the petrous pyramid involved only the periosteal layer of the bony capsule and spread into the dura and through the dura into the subarachnoidal spaces while the enchondral and endostal layers were but little if at all affected. This finding was more evident in the region of the cochlea and the vestibule than in the area of the semicircular canals, because the three layers of the bony capule of the inner ear are well separated from one another in the cochlea while they intermingle in the area of the semicircular canals. The process of bone resorption extended to the endosteum in several places in the semicircular canals, but even in these places the inflammation did not perforate the endosteum and thus did not carry the infection into the inner ear. The right temporal bone, therefore, presents the same conditions as Steinbrugge found except that in his case the temporal bone was invaded by a blood stream infection and not by direct extension, and that in his patient the labyrinthitis recovered spontaneously and the meninges were not invaded as in our case.

Osteomyelitis of the temporal bone involves only, or particularly, the periosteal bone of the middle ear as well as of the petrous pyramid. This is not surprising, since only this type of bone contains patent blood vessels even in old individuals.

While the right temporal bone of our case was affected by a direct extension of an osteomyelitis, the infection was carried into the left temporal bone by the blood stream. The left temporal bone also presents an acute purulent otitis media which, however, definitely differs from a common acute purulent otitis, both clinically and pathologically. From the clinical point of view it is noteworthy that the otitis media in osteomyelitis sets in insidiously, without pain, and that it does not produce marked changes of the drum, at least for a certain period of time. From the pathological point of view, two features of such an otitis are significant: *1.* It does not involve the entire mucous membrane of the tube, middle ear and antrum; it rather has a "spot-like" appearance. In our case the mucous membrane of the Eustachian tube was not involved, and that of the anterior

part of the middle ear was but little involved; on the other hand, the mucous membrane of the posterior part of the middle ear, particularly the region of the windows, was markedly affected. There was even more involvement of the mucous membrane of the antrum where the epithelium was destroyed in several places. Such a finding is very unusual. *2.* The inflamed mucous membrane soon activated the osteoclastic power of the periosteum and absorbed the adjacent bone on several sides. In our case even the footplate of the stapes showed deep Howship's lacunae without being entirely perforated. In the antrum the inflammation invaded the perantral pneumatic cells; it did not, however, extend far into the depth of the petrous pyramid, since the perilabyrinthine cells did not show any signs of definite inflammation.

It is not necessary to attribute the osteoclasis to a particularly virulent infection of the mucous membrane. One must bear in mind that the otitis occurred in an individual whose resistance was greatly diminished by the protracted course of the acute spreading osteomyelitis of the skull.

Although the changes in the middle ear on the left side certainly cause a marked diminution of hearing, these scarcely could produce the profound deafness of the patient. This symptom points to the cochlea as a further site of the disease. However, the findings in the inner ear were not conspicuous. There was no purulent labyrinthitis as found on the right side; there was no ectasia of the cochlear canal and a closure of the cochlear aqueduct found in "cholesteatoma deafness" reported by Siebenmann. In fact, there were no signs of inflammation. There was, however, a marked degeneration of Corti's organ, but it was not possible either to establish or to deny the intravital origin of these changes. Further, there was a definite atrophy of the spiral ganglion and the spiral nerve in the basilar portion of the cochlea. However, this finding also is insufficient to explain a profound deafness. The chief finding, therefore, is the interstitial neuritis of the cochlear nerve, characterized by a perivascular infiltration of the blood vessels within the cochlear nerve. This finding is not due to the meningitis found at autopsy, although in otitic meningitis the exudate frequently invades the nerves within the internal meatus on the healthy side. However,

under these circumstances all nerves of the internal meatus are more or less affected and there is either an equal infiltration of the entire length of the stem of the nerve or the pathologic process decreases toward the tractus foraminosus. In our case there was a "spot-like" infiltration of the cochlear nerve only.

We find that the profound deafness on the patient's right side was due to a purulent diffuse labyrinthitis and on the left side to a purulent acute otitis, an atrophy of the basilar coil of the cochlea, but perhaps chiefly to an interstitial neuritis of the cochlear nerve which probably was followed by a descending atrophy of Corti's organ. The findings on the left side, therefore, agree with those of Brock except that in his case there was a parenchymatous and not an interstitial neuritis of the cochlear nerve. One must realize that both the case reported by Brock, and the left side of our patient, present the early stage of a metastatic osteomyelitis of the temporal bone. Brock's patient became deaf 12 days, our patient only eight days before death. On the contrary, Steinbrugge's patient died four years after the onset of deafness. The question arises: From the pathological point of view, is there any relationship between Steinbrugge's case, on the one hand, and our case and Brock's on the other? At the first glance the answer must be, "No! There is no relationship between a purulent labyrinthitis and a cochlear neuritis." However, the state of affairs changes when one raises the hypothetical question: What would have occurred if our patient and Brock's had survived the onset of deafness for a longer period of time? There are two possibilities: *1.* It is possible that the acute otitis as well as the cochlear neuritis would have subsided. In such a circumstance the hearing would have been improved and there would have been no labyrinthine symptoms. *2.* It is possible that the destructive power of the acute otitis would have led to a perforation of the windows, as indicated in our case, and finally to a diffuse purulent labyrinthitis. In this instance no improvement of hearing would have taken place and there would have been no excitability of the labyrinth. The labyrinthitis, in turn, could either lead to a meningitis, or it could recover with the development of connective tissue and newly formed bone. By the latter assumption the finding of Steinbrugge is explained.

CONCLUSIONS.

1. In acute osteomyelitis of the skull the temporal bone may become infected either by continuity or by a blood stream infection.

2. In the former case, osteomyelitis involves only or particularly the periosteal bone of the external auditory canal, the middle ear and the petrous pyramid, inasmuch as only this type of bone contains patent blood vessels even in adults. From the periosteal layer of the petrous pyramid the infection may spread into the meninges.

3. If the infection is carried into the temporal bone by the blood stream, at first an acute purulent otitis media and an interstitial neuritis of the acoustic nerve may develop. This type of metastatic otitis media sets in insidiously without pain and does not produce marked changes of the drum, at least for a certain period of time. It does not involve the entire mucous membrane of the tube, middle ear and antrum; it rather has a "spot-like" appearance. The inflamed mucous membrane soon activates the osteoclastic power of the periosteum and absorbs the adjacent bone in several sites.

4. The deafness in acute, spreading osteomyelitis of the skull is due either to a diffuse purulent labyrinthitis or an interstitial neuritis of the cochlear and/or vestibular nerve, combined with an acute otitis.

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CANCER OF THE ESOPHAGUS: STUDY OF 100 CONSECUTIVE CASES.

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During 1940, carcinoma of the esophagus caused 2,804 deaths in the United States.[1] Based on per 100,000 population, its incidence has increased from 1.0 in 1915 and 1.7 in 1932[2] to 2.1 in 1940. It is seventh in frequency in types of carcinoma among males, exceeded only by carcinoma of the stomach, of the intestine and rectum, the prostate and bladder, the liver and pancreas, the lungs and pleura, and the buccal cavity and pharynx.

The present study concerns 100 consecutive cases, 80 of which were observed in the Research and Educational Hospital, University of Illinois, prior to July, 1942. There were approximately 137,000 admissions to the out-patient department during this period, or one patient with cancer of the esophagus to every 1,712 new admissions. Twenty additional cases were included from private sources, thus making a total of 100.

Age: As in all cancerous lesions, the primary cause is obscure. The secondary factors are of interest, however, and age is undoubtedly one of these. The youngest person in this series was 35 and the oldest was 83 years of age. There were five in the fourth decade, 11 in the fifth, 38 in the sixth, 34 in the seventh and 11 in the eighth, making the average age for the series 60.5 years (see Fig. 1).

Family History: A large number of the patients had been separated from their families in Europe and some had moved away from their homes in the Atlantic Coast States; therefore, in many cases no accurate history was obtainable as to the family background. The history of one patient showed that his father had died of carcinoma of the stomach; in a

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second case, the father had died of carcinoma of the bladder; and in a third, the father had developed carcinoma of the external ear following injury.

Sex, and Site of the Lesion: There is a remarkable variation between males and females in the site of neoplastic involvement. In women, carcinoma shows a distinct predilection for the postcricoid area. In six of the nine women in this series the growths occurred at this level. Of the remaining three, the tumor occurred in the middle third in one, the lower third in the second, and at the cardia in the third. Car-

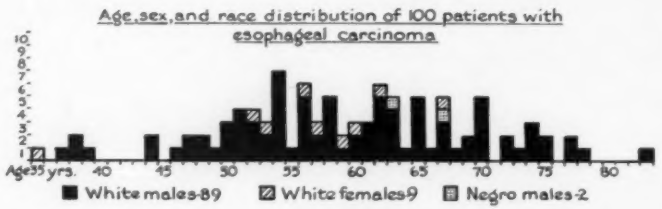

Fig. 1.

cinoma of the esophagus is primarily a disease of men, and the middle third is most frequently involved. There were 91 males in this series. The growths were distributed as follows: Three at the introitus, 22 in the upper, 39 in the middle, 14 in the lower third, and 13 at the cardia. In addition, there were three at the cardia which apparently had originated from gastric carcinoma. When the thoracic portion of the esophagus was involved, one could not determine the exact site of the original growth in an advanced case because of extension.

In 1940 in the United States 2,193 deaths from cancer of the esophagus among males were recorded as against 622 deaths among females. The proportion was 3.5 to 1. In our complete series this ratio is 9 to 1; however, exclusive of the private sources, there were only three women in the group of 80 treated at the Research and Educational Hospital, thus raising the proportion of men to women to nearly 27 to 1 in the low-income group (see Fig. 2).

Irritations: All writers on the subject agree that carcinoma of the esophagus bears a direct relation to the patient's mode of living and to the amount of irritation to which he is

exposed. Excesses in eating and drinking were frequent in these cases. The presence of poor oral hygiene and of dental sepsis was a striking feature in the patients. Among the 80 patients observed at Research and Educational Hospital, 27 were recorded as having either pyorrhea or dental caries, nine were using plates and only two had fairly good dental conditions. No records of the oral hygiene were made of the remaining 42. Contrary to popular belief, syphilis apparently plays no part in causation of the esophageal cancer, for there were only two patients with a positive blood Wassermann reaction. There were two with pulmonary tuberculosis; one

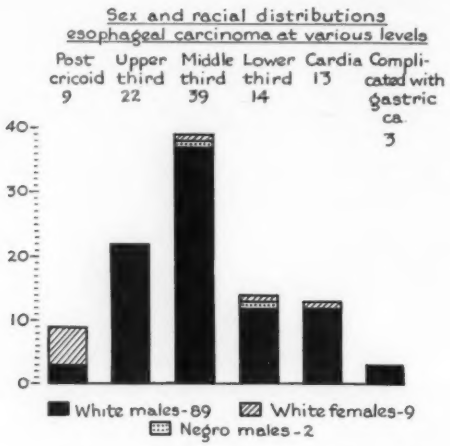

Fig. 2.

patient had pulmonary tuberculosis and a positive Wassermann, as well as esophageal carcinoma.

Alcohol and Tobacco: There were 21 patients who used moderate or excessive amounts of alcoholic beverages and 20 who smoked either cigars or cigarettes in excess significant enough to be mentioned in the history; however, specific mention of the use of tobacco was not made in the remaining 80.

Economic Status: More than half of those patients observed at the Research and Educational Hospital were foreign born and of lower economic levels. It has been suggested that hardship may be a predisposing factor in the development of car-

cinoma of the esophagus since most of the men in the group are accustomed to strenuous labor of various forms, but this does not warrant definite conclusions.

Race: There were only two negroes in this series. In the United States in 1940 there were 2,507 deaths from carcinoma of the esophagus among the white race and 198 deaths among all other races combined. The ration is 14 to 1.

Pre-Existing Scars: The histories as to previous illnesses were so varied that no definite conclusion could be drawn. Pre-existing scars were apparently the cause of carcinoma in two instances. Both were in women. The first patient was 67 years of age. Twenty-five years before the onset of her present illness she developed an organic stricture of the esophagus following typhoid fever. Numerous dilatations of the esophagus had been made. The neoplasm originated at the edge of the healed ulcer, most likely from a long continued irritation. The second woman was 52 years of age. She had had intermittent dysphagia over a period of 10 years, due to cricopharyngeal stenosis. The condition became gradually worse. For one and one-half years she had lived exclusively on a liquid diet, and two months before her admission to the hospital she developed an almost complete obstruction at the postcricoid area. One can scarcely believe that a malignant growth had existed 10 years. The fact that she had been in fair health for many years, and free from other symptoms that are peculiar to carcinoma, makes us feel that the tumor had its origin at the site of a benign stricture. A pre-existing diverticulum has been considered an important predisposing cause by some writers, but there was none in this series.

SIGNS AND SYMPTOMS.

Epigastric Pain: The absence of characteristic signs and symptoms in the earlier stage of the disease is most significant. The presence of the neoplasm initiates reflex irritations of distant regions, most frequently the epigastrium. Insidious onset without pain is the usual course. In 45 cases of our series, the epigastrium was indicated as the site of discomfort some time during the course of the disease. In all but two of these the lesion was located in the lower two-thirds of the esophagus, and in 12 epigastric distress was the initial symptom. The sensation was variously described as being a

feeling of fullness, discomfort, distress, slight pain, aching, heart burn, belching, soreness, dull pressure, boring, burning, cramping, shooting, cutting and gnawing. In the earlier stages, the epigastric symptom was intermittent and always associated with swallowing foods, and it ceased when swallowing was stopped. Later it became more or less continuous. In two cases an erroneous diagnosis of gastric ulcer had been made, and these patients had been placed on an ulcer diet seven months and eight months before admission to the hospital. In three cases the esophagus was secondarily involved in unrecognized gastric carcinoma. One of these, three months after the onset of the gastric disease, had had a partial thyroidectomy "in order to relieve the nervousness" in swallowing. In 21 cases, epigastric distress appeared soon after the onset of the initial symptoms of obstruction or almost simultaneously with it.

Substernal Pain: Substernal pain was the initial complaint of six patients, while in 23 this symptom appeared later. In six of these 29 patients the lesion was in the upper third; in 12 in the middle; in seven in the lower third; in eight the lesion was at the cardia, two being complications of primary gastric carcinoma. The sensation was described as that of oppression or weight beneath the sternum, or as a sense of tightness, of constriction and of cutting the chest, often radiating to the back or to the end of the xyphoid cartilage. This substernal discomfort is apparently caused by forceable dilatation of the narrowed area by the passage of a bolus of food and the accumulation of food in a slightly dilated portion of the esophagus, which in turn brings on increased muscular contractions. Several of the patients learned to relieve the distress by regurgitation which they voluntarily instituted. As in the epigastric pain, in the beginning the symptom was associated with swallowing food and was always intermittent; however, it always became progressively worse as the condition advanced.

Dyspnea and Hematemesis: One man noticed as his initial symptom that he became shortwinded on lifting heavy objects or when going up the stairs. This occurred two years before development of characteristic symptoms of obstruction. Hematemesis was recorded as the initial symptom in one case where the lesion was in the middle third of the

esophagus. In two cases it appeared later. Hematemesis is generally a later sign, and indicates ulceration.

Hoarseness: The altered character of the voice, huskiness or hoarseness was the initial symptom in five cases. So insidious was the change that one of the patients was totally unaware of the change in quality of his voice, though his family had recognized it for several weeks. In 20, hoarseness came later on in the course of the disease. At the time of their admission to the hospital, eight patients had recurrent laryngeal nerve paralysis on the left side and four on the right. In five others the paralysis was bilateral and in eight chronic laryngitis was present without paralysis. One of the men with bilateral paralysis showed a positive blood Wassermann reaction. He stated that his hoarseness had come on suddenly two months previously and that he had gradually become dyspneic. A tracheotomy was done soon after his admission. The paralysis of the recurrent laryngeal nerve is caused by pressure from the periesophageal nodes or from direct invasion of the nerve fibre by the neoplasm; it is almost always indicative of extraesophageal involvement. Hoarseness may result from constant overflow of the accumulated secretion into the larynx or it may be due to the local swelling and infiltration of the vocal cords without paralysis when the lesion is at the introitus.

Cough and Hiccough: Cough was recorded in 22 cases. It was usually worse at night and was annoying. In 14 cases the lesions were at the introitus or in its immediate neighborhood. In five, in the middle, and in three in the lower third of the esophagus. Cough was due either to overflow of the secretion into the lower air passage or from reflex irritation of the vagus nerve or its tributaries in the area adjacent to the site of the lesion. Persistent hiccoughs were complained of by two, due to the reflex irritation of the phrenic nerve or to a direct irritation of the diaphragm.

Sore Throats, Teeth Extraction and Tonsillectomy: Sore throat was recorded as the initial symptom in two cases. In a third, sore throat came on almost simultaneously with difficulty in swallowing. Because of the sense of discomfort in his throat, one patient had a tonsillectomy without benefit before admission. In all three the lesions were in the introitus. Numerous extractions of teeth were performed to relieve

unexplained discomfort in the region of the throat. Dull occipital headache was complained of as the initial symptom of another case.

Regurgitation: Regurgitation was recorded in 46 instances. In seven of these, the lesions were in the upper third and in 39 in the lower two-thirds. It was usually voluntary and occurred either during the course of a meal or immediately afterward when the esophagus was no longer able to retain the ingested food. Regurgitation is seldom delayed, and there is rarely as large a quantity regurgitated as in cardiospasm. Only in one instance was it recorded that regurgitation took place at the beginning of the following meal.

Aural Pain: Six patients found aural pain troublesome. The sensation was aggravated on swallowing. In one of these the lesion was at the introitus; in four it was in the lower two-thirds. The pain is due to reflex irritation of the tympanic branch of the Vth and IXth cranial nerves or the auricular branch of the Xth.

Appetite: Appetite remains good until very late in the disease. The sensations of hunger and thirst are seldom satisfied. Characteristically, patients stated that they felt nothing wrong except that they could not swallow and retain what they had eaten. At the later stage of the disease, as in all cancerous lesions, anorexia gradually sets in and the patients run a rapid, downward course, with cachexia, acidosis and terminal intercurrent diseases. In those complicated with gastric carcinoma, a loss of appetite, weakness and secondary anemia appear much earlier, so that the esophageal symptom is overshadowed.

Dysphagia: There were 51 patients who complained of dysphagia as the initial symptom. This was by far the largest single complaint registered in our survey. The sensation was variously described as a nervousness, a vague tightness, a slightly queer feeling in swallowing, the food sticking in the throat, or a lump in the chest. The vagueness of this complaint frequently led to an erroneous diagnosis of globus hystericus. The onset was slow and insidious, the difficulty being noticed first with solids, later with soft foods and finally with liquids. It was always intermittent at first. Only in one instance was it recorded that the dysphagia came on suddenly

as the patient was eating a meal in a hurry and became choked. The symptom had persisted until the end. The patients voluntarily selected a type of food which went down with greatest ease. They learned to be slow in eating, to masticate well, to take a smaller mouthful, and to add a large quantity of fluids to their food to wash it down and to avoid the inconveniences and embarrassment of regurgitation at the table. There were 40 who traced the onset of dysphagia to within four months of their admission. But the Roentgenographic and esophagoscopic evidences indicated far advanced lesions in all these cases. Dysphagia was noted earlier when the tumor had its origin at the postcricoid and hiatal levels. Jackson[3] states that dysphagia is almost always a late symptom. Mathews and Schnabel[4] state that 20.5 per cent of their autopsy cases showed no stenosis and were without the usual symptoms of obstruction. All of these growths were located in the lower two-thirds of the esophagus. Thus the clinical symptoms may be misleading. In our series, dysphagia was complained of by everyone some time in the course of his illness, although in some it appeared much later than in others.

Loss in Weight and Strength: Inanition is a primary cause of the loss in weight and strength observed. The largest single loss in weight was 120 pounds and the smallest was three pounds, while the average loss was 34 pounds. Gastrostomic feedings help to maintain normal weight and strength until very late in the disease.

Blood Pictures: The blood picture remains normal until toward the termination of the disease. Dehydration may concentrate the cell counts even above the normal level. Secondary anemia develops only in advanced cases. Reduction of red cell counts beyond 2,500,000 occurred only in those cases complicated by gastric carcinoma. Leucocytosis above 12,000 is indicative of the presence of secondary infections. Pleurisy, either with or without effusion, metastatic lung abscess, urinary retention with gangrenous cystitis, bronchopneumonia and lobar pneumonia were some of the complications which elevated the white cell count.

Carcinoma of the Tongue: In two patients the esophageal growth was apparently secondary to carcinoma of the tongue. One had had a partial resection of the tongue 20 years previ-

ously and had remained symptom-free until 12 months before admission to the Research and Educational Hospital. His chief complaints then were dysphagia and soreness of the side of the tongue. His esophagus was obstructed and there was a recurrence of the tumor of the tongue. The other had developed carcinoma of the tongue 10 months before admission to our service. In both instances the lesion was located in the midportion of the thoracic esophagus, probably from a direct implantation, and was completely separated from the lesion in the tongue.

DIAGNOSIS.

Roentgenology: Roentgenography is the most reliable, simple means of diagnosis at our command, though not without failures. The tumors at the introitus may be completely obscured by the bony structures and the laryngeal cartilages which undergo ossification in patients of cancer age. In this area, too, the strong muscles of the hypopharynx carry the barium through the cervical esophagus so quickly that small irregularities are difficult to outline. In four of our patients the Roentgenographic evidences remained uncertain for six to 10 months. Carcinoma of the cardia sometimes offers difficulty in diagnosis also. Cardiospasm, scarring with superimposed spasm, cicatricial strictures, diverticulum, non-malignant growth, and hiatal hernia with ulceration must be excluded. In two of our patients, an erroneous diagnosis of cardiospasm was made two months and six months before admission to the hospital. In each instance, a poorly made film was the contributary cause of the error.

Fluoroscopy with barium emulsion reveals the esophagus in action, and exposure is made in various positions. Even in the earliest stages in which the lesion is limited to a small area on the wall, defective folds of mucous lining can easily be delineated. A well made film with a contrast medium of proper consistency furnishes permanent objective evidence. Films made at later dates supply a means for comparative study of the course of the disease. Negative Roentgenologic findings in the presence of clinical symptoms demand a second and even a third Roentgenological study as well as an esophagoscopic examination.

Esophagoscopy: No method of examination approaches the

accuracy in diagnosis of the direct inspection of the lumen of the esophagus. It permits an exploration of every portion of the esophagus. A biopsy specimen gives indisputable evidence of the nature of the growth when examined by a competent pathologist. At times, more than one biopsy may be required before a final diagnosis can be made. In nine of the cases of this series, the lesion was an adenocarcinoma, some being papillary in type. All of these were located in the lower end of the esophagus. Three were extensions from a primary gastric carcinoma. There were 88 epidermoid carcinomas, one transitional cell carcinoma, one basal cell carcinoma and one colloidal carcinoma. The majority of the epidermoid carcinomas in this series showed marked virulency. No tumor was found which could be classified as grade I according to Broder's classification. A few cases were classified as grade A. By far the largest number of cases was classified as grades 2 and 3. The esophagoscopic appearance of the growth depends on the site and stage of development. In the earlier stage, a direct examination may reveal only an area of slight induration with a possible suggestion of narrowing of the lumen. In an advanced stage, the endoesophageal lesion usually presents an ulcerative, verrucous, pedunculated, polypoid, fungating, cauliflower-like mass covered with thin, dirty, yellowish exudate, often filling the entire lumen. A mere touch with the tube mouth frequently brings on profuse bleeding. Such a lesion prevents the examiner from viewing the entire extent of the growth. He sees only its proximal portion. This form of tumor constituted a large proportion of our cases. In the hard, fibrous, scirrhous type of growth, the tumor tends to grow excentrically and later encircle the wall of the gut. The intrinsic, endoesophageal growth is relatively small and does not encroach on the lumen. The esophageal mucosa remains intact until very late. The periesophageal portion may attain a considerable size impairing the motility of the esophagus. Such a lesion may be three or four inches in length. In an early stage, biopsy may or may not be obtainable, but the esophagoscopic appearance of the tumor is so characteristic that it is almost unmistakable. According to Mathews and Schnabel,[4] there is a third type of esophageal growth which causes no stenosis. This constituted 20 per cent of their autopsy material. The growth presents a shallow ulceration. None of our cases was of this type.

Fig. 3 indicates the time between the onset of the initial symptom and biopsy. The shortest was two weeks and the longest 10 years, the lesion in the latter beginning as a cricopharyngeal stenosis. Owing to mental derangement in one patient, no history was obtainable. Forty-three per cent of the cases reported an interval of less than four months and 62 per cent less than six months between the initial symptom and the time of biopsy.

COMPLICATIONS.

Complications: To understand various complications that are met with in this disease, one needs but to review the

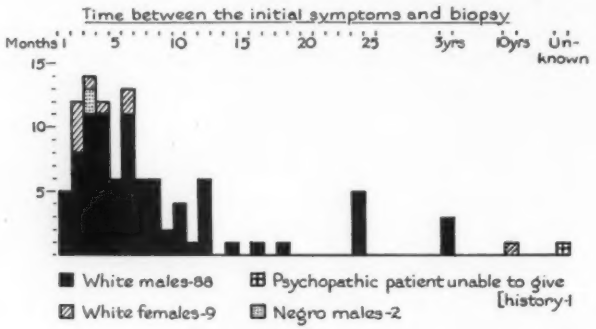

Fig. 3.

anatomy of the region and the mode of extension of the growth. The larynx and the trachea lie in front of the esophagus at its upper third. The carotid artery, the recurrent laryngeal nerve and the pleura are in close proximity. In the middle third, the trachea, the left bronchus, aorta, left subclavian artery, intercostal arteries, hemiazygos veins, thoracic duct and the pleura are in immediate contact. In the lower third the left auricle, pericardium and the descending aorta lie immediately adjacent to the esophagus. The esophagus is richly supplied with lymphatics. Two distinct layers of reticular network embrace the submucosa and muscularis. The periesophageal glands cover the entire length of the gut. Those from the upper half drain into the posterior mediastinal, the deep cervical and paratracheal nodes. In two cases of our series, in which carcinoma was in the upper esophagus,

there was an extensive involvement of the soft tissues of the neck. The lymphatic glands from the lower half of the esophagus drain into the posterior mediastinal, paracardial and subdiaphragmatic nodes along the walls of the stomach and the liver. In six patients of our series the liver was palpable at the time of their admission to the hospital for diagnost study. The spread of the growth takes place *1.* by direct extension, *2.* by lymph-vascular channels, and *3.* by implantation. In rare instances carcinoma may invade the venous streams to produce thrombophlebitis and cause widespread metastases in the lungs and other distant organs. It has already been mentioned that the recurrent laryngeal nerves are the first and the most frequent to suffer. Respiratory complications encountered in this series included pleural effusion, bronchopneumonia, suppurative pneumonia, pulmonary abscess, empyema, tracheobronchial invasion and fistulas. They were the causes of death in the majority of our cases. Fatal hemorrhage occurred in one. Souttar[5] has stated that of every 10 cases of carcinoma of the esophagus, nine die from purulent pneumonia and one from hemorrhage. Tracheotomies were done in seven of our cases.

Because most patients lived at a distance and were discharged after the initial treatments, we have no available data as to the average length of life after a diagnosis was made. In a series of 297 cases reported by Nathanson and Welch,[6] the median life expectancy of the untreated patients was found to be seven months; those treated with irradiation, 9.3 months; and those who received gastrostomy, 10.4 months.

Autopsy: There were 13 institutional deaths and all came to autopsy. The scarcity of autopsy material is largely due to the planned program which is in practice in many State and County institutions, eliminating those with long and lingering illnesses to make room for more acutely ill patients. Tracheobronchoesophageal fistulas had occurred in five; bronchopneumonia in eight; and lung abscess in two. In eight, the growths had either extended to the periesophageal nodes or were limited to the wall of the esophagus. One patient died of massive esophageal hemorrhage, and another of gangrenous cystitis from urinary retention. In one a secondary deposit of multiple tumor masses on the mucous membrane of the esophagus had occurred some distance from the pri-

mary growth, leaving healthy mucosa between the two sites. In five, there were metastases to distant organs, chiefly to the liver, stomach and lung. The incidence of metastases was 36.5 per cent. Ochsner and DeBakey[7] in their exhaustive study on the subject have shown that metastases occurred in 418 instances among 1,025 collected cases that came to autopsy, or an incidence of 40.7 per cent. Jackson[3] states that esophageal carcinoma long remains a localized disease and metastases occur only when the growth extends beyond the confines of the protective wall. There is a growing volume of opinion that the tumor is highly virulent and that most of the patients succumb from its effects before metastases occur in distant organs. This tendency for localization of cancer of the esophagus is of great significance for a successful extirpation of the diseased portion and an ultimate cure.

TREATMENT.

The treatment is both palliative and curative. The palliative measures include *1.* medical; *2.* intubation; *3.* dilatation; *4.* protracted or fractional irradiation; *5.* gold-filtered radon seed implantation and intraesophageal radium element tandom; and *6.* gastrostomy. The curative measures include *1.* removal of the growth by diathermy, cautery, and *2.* surgical excision.

Dangers of acidosis, dehydration, mental depression, undue fatigue and loss of strength must be constantly guarded against. Foods must not only be adequate and appetizing but also nonirritating to the growth as they pass down through tortuous channels of the growth. In a selected few, an intubation may be tried. Two of our cases were so treated.

A gentle dilatation of the involved portion under direct vision or by bougies passed over a string is a useful procedure. The fact that a number of our patients voluntarily returned to the clinic for this type of treatment speaks well for its value; however, the danger of esophageal perforation cannot be minimized. In 14 patients, gold-filtered radon seeds were inserted directly into the growth. In 22, protracted fractional irradiations were carried out according to the radio sensitivity of the growth and the individual tolerance.

Voluminous literature, both favorable and unfavorable, has

appeared, dealing with the subject of gastrostomy for patients with carcinoma of the esophagus. Fifty-one of our patients either received gastrostomy after admission to the hospital or had had the operation performed before entrance. Four were advised to have it, but refused the operation. We believe that in a well selected case, an early gastrostomy is free from operative risk and is the most satisfactory treatment. It affords an avenue for supplying an adequate amount of fluid and balanced nutrition, and at the same time relieves the esophagus from exposure to constant irritation by ingestion and stagnation of foods.

Moersch[8] reported a case in which a small intraesophageal growth was removed by surgical diathermy. There was none in our series in which such treatment was feasible. In two, a transthoracic extirpation was undertaken. In one, at the time of gastrostomy a subdiaphragmatic metastasis was discovered and the plan was abandoned. In the second case a neoplasm in the distal portion of the thoracic esophagus was successfully dealt with in April of this year by removal of this segment and the patient is living and well.

The encouraging reports[9] in the literature lead to the opinion that surgical treatment of carcinoma of the esophagus is not only within the realm of possibility but is readily feasible and the only curative method of management of the disease available. Thus, it is imperative that those who see these cases in their early stages of development be cancer-conscious and be ever on guard for an early recognition. Only in this manner can we reduce the present frightfully high mortality rate from this disease.

SUMMARY.

These studies corroborate the prevailing impression that cancer of the esophagus is a disease of men. The white race is much more prone to develop it than the colored races. Heredity appears to play no part. Poor oral hygiene, excesses in eating and drinking, alcohol and tobacco are suspected, although it is difficult to establish them as contributory factors. Carcinoma of the esophagus is a highly malignant disease. Because of its insidious onset and relatively smooth course in its incipiency, no patient can definitely establish its beginning. The tumor remains localized until very late and

metastases to distant organs are found in less than 40 per cent of the cases. Pre-existing benign growth, scars, diverticula, hiatal hernia, cardiospasm and ulcer are thought to be predisposing factors.

X-ray and esophagoscopy are the two most important factors in establishing the diagnosis. Dysphagia is always intermittent in the beginning but is usually persistent as a late symptom. It is indicative of obstruction due to endoesophageal growth or infiltration of the wall of the esophagus, interfering with its normal function. Absence of demonstrable physical signs is the characteristic feature of the disease. An erroneous diagnosis of globus hystericus, cardiospasm or gastric ulcer is apt to be made in the early stages. There is no correlation found in this series between the patient's complaints and the extent of the lesion. Surgical treatment offers the best opportunity for cure while the growth is still limited to the lumen of the esophagus.

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700 North Michigan Avenue.

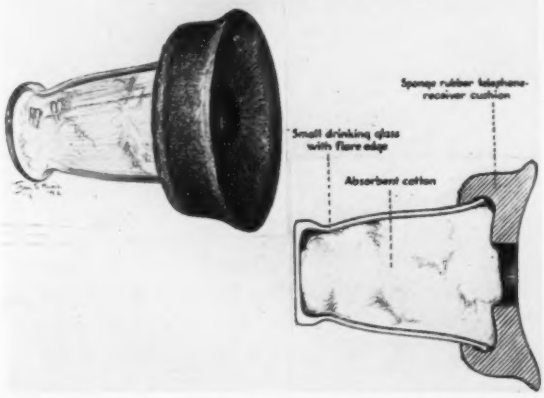
PRESENTATION OF A SIMPLE DEVICE FOR EXCLUDING INTERFERING NOISES FROM ONE EAR WHILE TESTING HEARING ACUITY OF THE OTHER EAR.*

DR. HORACE NEWHART, Minneapolis.

On the gracious invitation of President Seydell, in order to conserve time, I take this opportunity to present, a simple, practical device which I recently have improvised and have found effective when testing the hearing of children and adults in school and office buildings where the prevailing noises interfere with obtaining accurate results. It has proved useful when testing by the pure tone audiometer, the acoumeter, watch, whisper and softly spoken voice.

The device consists of a small fruit juice glass loosely packed with absorbent cotton, and a standard Trimm No. 660 soft sponge-rubber telephone receiver ear cushion which tightly fits the flaring rim of the glass, as illustrated.

Device for excluding interfering noises from one ear while opposite ear is being tested for hearing



The glass serves as a convenient handle for the device and the cotton packing absorbs the reverberations of such noises as penetrate the glass. The soft sponge-rubber receiver ear cushion held over the auricle with moderate pressure against the head acts as a gasket and reduces the level of interfering noises to a practical minimum.

By actual test the transmission loss obtained is from 15 to 35 or 40 dcb., according to frequency. This inexpensive device we have found more cleanly and more uniformly effective than the time-honored use of the finger inserted into the meatus. The psychological effect is excellent in testing the imaginative, hypersensitive child or adult.

Unfortunately at this date, owing to Government priorities, the sponge-rubber receiver cushions are off the market.

527 Medical Arts Building.

*Presented at the Seventy-fifth Annual Meeting of the American Otological Society, Inc., Atlantic City, May 29, 1942.

HEMATOLOGY.

DR. JOHN J. SHEA, Memphis.

Hematology is an important science to the laryngologist, for in his field of endeavor many of the hematopoietic diseases have their initial manifestation. The older physiologist considered blood as a special form of connective tissue with a fluid matrix and a twofold office; first, to carry to the tissue materials for their nourishment, and second, to take away from the tissues the by-products of metabolism. Today, blood is considered a mixed secretion, whose formed elements are the results of parent cells living outside of the blood stream and whose functions include oxygen transported by the red cells, osmotic pressure maintenance by the plasma proteins, coagulation through the prothrombin-fibrinogen mechanism, nutrition with special reference to the rôle of the plasma proteins, participation in body response to infection through complement and circulating antibodies, and phagocytosis by the leukocytes.[1]

The Archives of Internal Medicine have reviewed hundreds of articles written on blood. The 1941 review was edited by S. Milton Goldhamer, Cyrus C. Sturgis and Frank H. Bethell[2] and the 1942 review was edited by Frank H. Bethell, Cyrus C. Sturgis, Robert A. Hettig and Otto Todd Mallery.[3] To these belong a great credit for making possible this presentation and to them the reviewer gives full credit for many of the ideas in this article.

TRANSFUSIONS.

The value of stored plasma in large quantities was graphically illustrated in the care of the wounded at Pearl Harbor and to one of our own laryngologists, Dr. Forrest J. Pinkerton, must go credit for possessing the foresight to have had a blood bank established and capable of functioning in Honolulu.

When possible, whole blood should be administered to replace blood that has been lost. The employment of whole blood is rarely contraindicated except in instances of extreme

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hemoconcentration, as may occur following severe burns. Plasma and serum have the advantage, particularly in emergencies, that compatibility tests are not necessary and delay in treatment can be avoided. Preliminary observations indicate that purified human albumin is a safe and effective substitute for plasma. The problem of providing blood substitutes in large quantities will be greatly simplified if nonantigenic fractions of animal plasma proteins can be prepared.[4] The administration of plasma has the advantage of being moderately safe regardless of the blood group, because isoantibodies will be diluted sufficiently to prevent agglutination.

Levine and State[5] call attention to cutaneous tests for the presence in plasma of A and B factors, they being the same antigens as occurring in the erythrocytes of A and B bloods. This sensitivity is not specific to any special blood group and their experiments conclusively prove that there are no reactions following the intravenous administration of plasma, which gave a negative cutaneous test, and advise cutaneous testing as a valuable adjunct to plasma therapy.

ANEMIAS.

In 1926 when Minot and Murphy announced that liver was effective in the treatment of pernicious anemias, they opened a new approach in hematology. Castle subsequently discovered that in order to maintain the normal blood picture there must be an intrinsic factor which is present in the stomach normally but absent in pernicious anemia, and an extrinsic factor obtained from foods such as liver. But the stomach of a pernicious anemia could never digest the amount of liver necessary to be taken by mouth. That would not be possible. The Armour Co. pioneered in the preparation of extracts capable of being given hypodermatically.

Bethell[6] advised 15 U.S.P. units daily for one week and subsequently three times a week until the blood pictures are normal, then a maintenance dose of 15 units once a week or twice a month is sufficient to maintain 5,000,000 R.B.C. and a hemoglobin concentration of 100 per cent. Oral liver therapy is only a substitute method and unreliable.

During the present war the supply of fresh liver to a per-

nicious anemia patient in England was found unnecessary, except for those who were sensitive to the injections of liver extract.[7]

Turner[8] reported three cases of optic atrophy, the result of pernicious anemia, and recommended gastric and blood analysis in patients with bilateral optic atrophy of middle life and suggested the use of liver extract in their treatment.

Craddock[9] reported two deaths of eight cases of hemolytic anemia following the administration of sulfanilamide and the recovery of one case the result of sulfapyridine. The renal lesions were similar to those seen after transfusion reactions.

INFECTIOUS MONONUCLEOSIS (BENIGN LYMPHADENOSIS).

Pfeiffer[10] established the existence of the disease as a clinical entity and gave it the name of glandular fever in 1889. Subsequent observers agree that the disease is infectious and numerous epidemics have been reported. The frequency of outbreaks in our universities, colleges and high schools of an acute infection of the throat, complicated by glandular enlargements, has attracted attention to infectious mononucleosis. Sporadic cases in adults occur, but are missed, because of the failure to have routine blood studies made in our office practice.

The etiology is that of a virus, as considered by Barber,[11] whose three patients had ulcerations of the tonsils, resistant to treatment. Sporadic cases usually occur in adults, while epidemics occur among children. The incubation period is from five to 15 days. There are two views regarding the cause of the response of the blood, one that a specific infective agent gives rise to the characteristic lymphocytic response; the second, that the lymphatic reaction is probably due to an unusual constitutional disposition on the part of the patient reacting to an ordinary acute infection.

Grove[12] presented the study of nine cases to promote a closer co-operation between the laryngologist and the serologist. The disease may be confused with diphtheria, mumps, follicular tonsillitis, streptococci pharyngitis or sinusitis, accompanied by cervical adenitis. In children, the leukocyte count may reach its maximum at the end of the first week, whereas in an adult more time is required. Headache is a

common symptom in adults, though the number of cells in the spinal fluid will average less than 100 per cmm.

Kaufman[13] contends that 2 to 10 per cent of infectious mononucleosis patients will give a false Wassermann reaction and suggests that in the laboratory procedures, false positive reactions will be checked by the heterophile antibody test. The cause of the false positive reaction in infectious mononucleosis is unknown. It usually appears during the second week and may persist for several months and affects flocculation procedures, as the Kahn test or the Wassermann reaction.

SYMPTOM.

The onset is gradual, the patient complaining of fatigue, dull headache, general malaise followed by fever, sore throat and lymphadenopathy of the cervical, maxillary and inguinal regions.

The physical examination reveals an elevation of temperature and an enlargement of the spleen, and an angioma with an enlargement of the tonsils and cervical lymph glands. Suppuration seldom occurs, unless complicated by a mixed infection. The posterior cervical lymphatic chain enlarges and sometimes the deeper chain under the sternocleidomastoid muscle. The clinical importance of a diagnosis is the mental relief, that the individual has a reason for the protracted disease of what appeared at first to be a simple tonsillitis or to the exclusion of a more serious diagnosis, such as acute lymphatic leukemia or Hodgkins disease.

HEMATOLYTIC FINDINGS.

The red blood cells and the hemaglobin remain normal or show a late secondary anemia. The total white count is not typical. If made early, it will reveal a leukopenia from 3,000 to 4,000 cells and as the glands enlarge it increases to 20,000 or more; 50,000 cells suggest an acute leukemia.

The hemogram displays a shift towards an increase in mononuclear cells at the expense of the polymorphonuclear neutrophils. The mononuclear cells include both monocytes and lymphocytes. The former, in normal numbers, are slightly increased, while the lymphocytes show a relative and abso-

lute increase of 50 per cent or more. The abnormal blood picture may persist for months after the initial attack.

DIAGNOSIS.

The septic cases must be differentiated from pyogenic septicemia, acute leukemia, acute Hodgkins disease and the acute fevers characterized by lymphocytic hemic responses.

The heterophile antibody test is diagnostic when positive in the higher titers. Any dilution above 1:64 is considered diagnostic if there is no history of serum sickness. The titer seems to be higher when the temperature is higher and remains elevated for months. The prognosis is favorable, complications are rare and fatalities depend upon secondary infections.

TREATMENT.

The symptomatic treatment of the anginose type of infectious mononucleosis may be either refrigeration of the cervical glands by the use of cold compresses or the application of continuous heat; *i.e.*, antiphlogistine packs and the daily use of radiation by the water-cooled ultra violet will remove the tenderness and some of the swelling. Many clinicians advocate deep Roentgen therapy when the former fails or the glands involved belong to the deeper chain. Transfusions of convalescing serum assist in building up an immunity to the offending virus. Saltzman[14] advises the intramuscular injection of 0.03 mg. of bismuth and potassium tartrate. Whether this pharmacological compound has a specific action or its effect is due to increasing the number of polymorphonuclear neutrophils will require confirmation. Maintenance of thorough dental hygiene when administering bismuth therapy has been found to be a good practice, as the metal possesses a tendency to deposit about the teeth.

Thomsen[15] reported gratifying results in the treatment of infectious mononucleosis with sulfanilamide and convalescent serum. He contends that sulfanilamide alone exerts its beneficial effects upon the secondary invading organism.

HOWELL'S THEORY OF COAGULATION.

Adequate ionized calcium, thromboplastin and fibrinogen must be present for the normal clotting of blood. When the

thromboplastin is liberated by tissue injury in the presence of vitamin K, it is converted into prothrombin, which when combined with calcium forms thrombin, a fibrin ferment capable of converting the fibrinogen into fibrin. Vitamin K is a product of alfalfa and K^2 is obtained from putrefied fish-meal. It is accepted that prothrombin is a product of liver activity and that the absorption of vitamin K is favored by the presence of bile salts. Synthetical vitamin K substances are capable by oral administration of re-establishing normal clotting time, when the deficiency is due to a lack of prothrombin.[16]

The use of one of the sulfonamides as a dusting powder in operating wounds promotes a moderate oozing. Cunningham,[17] in the June issue of *Annals*, states "the application of powdered sulfapyridine and sulfamethylthiazole to experimental wounds in the horse, guinea pig and rabbit result uniformly in the control of vascular oozing. These results confirm observations made in cases of delayed and secondary tonsillar hemorrhage in which the aforementioned drugs were used successfully in the control of persistent bleeding."

This happy promise of aid in the controlling of persistent vascular oozing of secondary tonsillar bleeding has not materialized in our experiences.

AGRANULOCYTOSIS (MALIGNANT NEUTROPENIA).

Though Gubler,[18] in 1857, and Trousseau,[19] in 1865, described an angina similar to agranulocytosis, credit must be given to Schultz[20] for the term "agranulocytosis." Bethell advises the study of Plum's[21] work on agranulocytosis as the most authoritative and comprehensive discussion available.

ETIOLOGY.

Bacterial: Numerous observers agreed with Lovett[22] that the bacillus pyocyaneus, because of its ability to produce leukopenia, was the offending bacteria, later the frequency with which the spirochaetes of Vincent's and its associate, the fusiformi bacillus, were isolated from the mouths of patients suffering from agranulocytosis brought these organisms under suspicion. There is no doubt that some cases are the result of severe sepsis, but no organism has consistently been associated with the disease.

Chemical: Jackson[23] personally collected 109 instances of the condition following the administration of drugs: Sulfanilamide, 34; sulfapyridine, eight; aminopyrine, 39; allonal, seven; causalin, four; and accused cibalgine, acetanalid, amidophen, cinchopen, amytol compound, bismarsen and neocinchopen. Thompson[24] reported a fatal case following sulfathiazol. Curry[25] reported an acute agranulocytosis occurring after sulfadiazine therapy. Leser[26] cites the work of Peper, Long and Bliss, that no instance of acute agranulocytosis has followed sulfathiazol, but Kennedy and Findland[27] reported a fatal case folowing the administration of sulfathiazol. Acute hemolytic anemia may develop in the first 24 hours, but it requires two weeks' administration of the drug before the onset of agranulocytosis. Pippin[28] emphasizes ominous importance of fever following the use of sulfathiazol. Kracke[29] observed that eight out of nine patients seen by him with acute agranulocytosis had received coal tar products and suggested that the benzene ring was of etiological importance. Arsphenamine, bismuth, gold and benzene when improperly used are capable of producing a neutropenia.

PATHOLOGY.

The ulcerations occur most frequently in the mouth and about the anus or in the vagina; in these areas where bacteria are constantly present. The lesion is a round cell infiltration and characterized by the absence of polymorphonuclear neutrophils. The exciting cause may be bacterial or chemical, but in either case it is the myeloid elements of the bone marrow that is affected. The severe reduction of the neutrophils is the result of their destruction in the peripheral blood stream or their migration into the viscera or the failure of maturation of the myeloid elements in the bone marrow. There is an unknown X factor in hemopoiesis which is a barrier that prevents immature cells from being dumped into the peripheral circulation. Perhaps in this disease the barrier prevents the normal discharge of mature granulocytes. There is little evidence to support the destruction theory and the idea of abnormal distribution lacks verification as the blood obtained from the organs varies slightly from that of the peripheral circulation; apparently, then, it is a failure of maturation. Occasionally the shift occurs before a lesion can be isolated. Jackson and Parker[30] reported a case

in which this occurred five days before the sore throat was visible. The reviewer observed a fatal case for three days before the appearance of a lesion, whose blood was typical of agranulocytosis.

TREATMENT.

The local care of the ulcerations should be limited to mild oxidizing agents and the avoidance of caustics. At times the lesion about the tonsil will imitate an early peritonsillar abscess. Surgery should be avoided, and even aspiration is dangerous. Administration of vitamins is of temporary advantage, especially nicotinamide. Transfusions are temporary measures and, as the blood is expendable, must be repeated according to the demands. Watkins[31] advocated the administration of yellow bone marrow concentrate for several days. Pentnucleotide 40 cc. a day is the most potent therapy. Conservative application of Roentgen therapy was recommended by Dowdy,[32] Murphy[33] and Lawrence.[34]

Leser[35] advises intensive parental liver therapy with repeated blood transfusion and 10 cc. pentnucleotide, four times a day. Davis and Wingfield[36] utilized injections of epinephrine to increase the polymorphonuclears when pentnucleotide therapy and blood transfusions failed.

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1018 Madison Avenue.

SUFFOCATION BY A SUNFLOWER SEED.*

DR. GOODLATTE B. GILMORE and DR. MAURICE D. GRANT,
New York.

The Jacksons[1] report an instance of suffocation by an oyster straddling the carina and blocking both main bronchi. Such accidents from an aspirated foreign body are rare.

R. E., a 2½ year old white female, was admitted to Morrisania City Hospital on Oct. 7, 1941, at 4:30 P.M. Three days before, a cough mixture was prescribed for a "cold." On Oct. 6 the patient had "difficulty in catching her breath for a few minutes." On the morning of admission, respiratory distress returned and was present on entering the hospital. No history suggestive of an aspirated foreign body could be obtained at this time.

Examination revealed a well developed child with marked laryngeal dyspnea. On inspiration, retraction at the suprasternal, infrasternal, supraclavicular and intercostal spaces was noted. Examination of the chest was negative. Diffuse edema of the larynx and redness of the pharynx were observed. The temperature was 102° F., pulse 150 and respirations 33 per minute.

A diagnosis of acute laryngotracheobronchitis was made and a tracheotomy was done immediately.

Following the operation the dyspnea disappeared. The child sat up, drank some water and even smiled. Neoprontosil (25 cc., 5 per cent) was given intramuscularly and an infusion of 5 per cent glucose in saline solution was started. The humidity in the room was elevated with steam.

A white blood count done at this time was 16,100, with 90 per cent polymorphonuclears, 8 per cent lymphocytes and 2 per cent transitionals.

Three hours after the tracheotomy the child stopped breathing. The tube was patent and suction applied in the trachea was nonproductive. Death came quickly.

At autopsy a sunflower or "polly" seed, situated obliquely in the trachea at the level of the carina, was found. One end of the foreign body had become impacted in the orifice of the left main bronchus (see Fig. 1). There were redness and swelling of the tracheal mucosa but purulent secretion was not abundant.

The streptococcus hemolyticus and staphylococcus albus grew in cultures taken from the pharynx, larynx and trachea at the time of operation.

When informed of the autopsy findings, the mother remembered the child had played with sunflower seeds on the first day of her illness, but she denied that there had been any coughing or choking at this time.

COMMENT.

It can be assumed that the foreign body was freely movable in the trachea for three days without producing an

*From the Otolaryngologic Department of Morrisania City Hospital.

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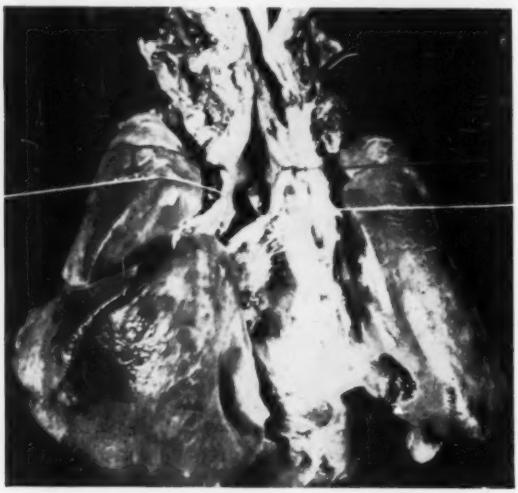
"audible slap" or "asthmatoid wheeze" (Jackson), recognized as such. During this interval a vegetal laryngitis and tracheitis developed. Subsequent to the tracheotomy, the seed changed its position and because of the swollen mucosa obstructed both main bronchi at the level of the carina. Had



a bronchoscopy been performed prior to tracheotomy, the foreign body might have been seen and removed.

This case illustrates the difficulty of making a diagnosis in obstruction caused by vegetal foreign bodies and emphasizes the extreme importance of bronchoscopic examination either prior to or immediately following tracheotomy in all such cases.

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2940 Grand Concourse.

1860 Morris Avenue.

IN MEMORIAM

EWING W. DAY, M.D.,

1862-1942.

It is with extreme regret that we record the death of Dr. Ewing W. Day, of Pittsburgh, Pa., and Provincetown, Mass., one of the staunch supporters of otolaryngology. He passed away, Nov. 25, at Doctors' Hospital in New York, at the age of 80 years.

Dr. Day belonged to that group of specialists who contributed much toward the establishment and development of otolaryngology in this country.

Born in Deerfield, Ohio, the son of the late Edgar M. and Frances Reed Day, he graduated from Allegheny College, studied medicine at Georgetown University and took postgraduate work in medicine in Vienna.

He was Past President and one of the founders of the Academy of Medicine in Pittsburgh, and Past President of the American Otological Society, Inc., the American Laryngological, Rhinological and Otological Society, and the Allegheny County Medical Society. He was a member of the International Collegium Oto-Rhino-laryngologicum.

Dr. Day was a man of striking and positive personality; he was frank, courageous, cheerful, friendly and cultured. His cordial greeting and evident pleasure in meeting and exchanging ideas with his medical colleagues was one of the refreshing moments at otolaryngological meetings.

To his talents for organization and administration can be traced much of the success of the various otolaryngological societies.

His last years were spent in the peaceful environs of Provincetown, Mass., where his pleasant home and his beautiful garden gave him many hours of relaxation, interspersed with regular attendance at medical meetings where he enjoyed the contact with his former colleagues. He will be greatly missed by his friends in the profession.

He leaves his widow, Mrs. Annie Mosier Day, three sons, Dr. Kenneth M. Day, a practicing otolaryngologist of Pittsburgh, Percival E. Day and Maj. Ewing W. Day, II, and two grandchildren, to all of whom we extend sincere and heartfelt sympathy.

T. E. W.

JOSEPH C. BECK,

1870-1942.

JOSEPH C. BECK, M.D.,

1870-1942.

Joseph C. Beck was born in Dobris, Czecho-Slovakia, on Sept. 26, 1870. He attended public schools in Prague up to the age of 14 years, at which time he came to America. He attended parochial schools in Holden, Mo. After graduating from the University of Illinois, he spent 18 months in the clinics of Europe. He began the practice of otolaryngology in Chicago in 1901. He was a Fellow of the American Otological Society, American Laryngological Association, American Laryngological, Rhinological and Otological Society, American Academy of Ophthalmology and Otolaryngology, American Bronchoscopic Society, American Medical Association, American College of Surgeons, Member of the Chicago Medical Society, Chicago Otological and Laryngological Society, Chicago Ophthalmological Society, Chicago Medical Historical Society, Beck's Reading Club of Otolaryngology.

Associate Professor of Otolaryngology, University of Illinois College of Medicine.

Chief Otolaryngologist, North Chicago Hospital; Attending Otolaryngologist 17 years under Civil Service, Cook County Hospital; Senior Attending Otolaryngologist, Michael Reese Hospital; Consultant in Otolaryngology, Hines Veterans Hospital.

Member of Board of Directors, American Board of Otolaryngology, 1925—; Chairman, Section on Laryngology, Otology and Rhinology, American Medical Association, 1919; President, American Academy of Ophthalmology and Otolaryngology, 1915.

Major, M. C., 1918; Lieutenant-Colonel, 1919—.

Served overseas with the French Division of the Czecho-Slovaks in Cogniac, France, winter of 1918, as Lieutenant-Colonel and in charge of American Red Cross Hospital 113.

Dr. Beck died in Chicago, Oct. 20, 1942. He is survived by his widow, his daughter, Miss Elsa Beck, and two sons, Joseph C. Beck, Jr., and Philip Beck.

Memories of Joe Beck are pleasant particularly to me because I knew him intimately longer than anyone else I know. Thirty-two years ago when we were both young, he

visited our home in Iowa City. He was a perfect guest; he was considerate of everyone. Our servants were Bohemians and Joe felt perfectly at home. He spent most of his time visiting the university, always spending his time in the departments of anatomy and pathology. These were things he was interested in. I think his broad knowledge of pathology when a young man had much to do with his success.

Joe was nice with children. I remember when he was at our home 32 years ago that he and our son were good chums. One day we were sitting on the west porch and the boy brought Joe a drink and some carbonated water. He called the boy "bubbly water" and through the years he remained "bubbly water" to him. It made no difference whether I met Joe in a business meeting or at a cocktail party, he always asked about "bubbly water." A short time before his death, when he was too ill to see us, he asked about "bubbly water." This is indeed a very small matter but it was the summation of many such things that made him very human and a great man.

As a young man he was a tireless worker. He enjoyed operating before an audience, teaching all the time. I personally received much benefit from watching him. He was never nervous but once: He was doing a nasal operation under local anesthesia and it was evident that he was disturbed about something. He twisted around in his chair and did not begin the operation. Finally he said to the patient, "What have you in your pocket?" "Oh," said the patient, "only my gun," and he pulled out of his pocket a 32-revolver. Joe immediately became energetic and said, "Put that thing over in the far corner of the room. I won't operate anyone with a thing like that in his pocket."

He was kind in handling his patients being operated on under local anesthesia. Women and children he talked to all the time and kept them under perfect control.

Joe was a big city man. Away from the big city, his judgment was not so good. Thirty years ago Mrs. Dean and I were in the backwoods of Minnesota where log-cutting was in progress. One day a timber cruiser brought us a telegram saying Joe would arrive at a logging station 10 miles away.

When we received the telegram he was already there so we started for the station. It was fortunate we were at the cottage when the message arrived. When we reached the station we found Joe not looking for us but waiting for the first train to get him out of what he called a "God forsaken" place. We did our best to get him to go to our cottage but he had had enough.

On his arrival he noticed a frame shack labeled "hotel." He went there and found only one man in the office, an unshaved giant in a red shirt and big boots, who pulled a bottle out of his hip pocket and offered Joe a drink, which he politely refused. The man went over to the corner of the room and picked up a double-edged timber ax. Then he said, "Now will you drink?" — and Joe drank. Then the man left and soon a second person arrived. Joe asked him if the cook was in the house. The man told him that he was cook, chambermaid and owner of the place.

Joe did not dare refuse to eat the meal that was placed before him and escaped to the station where we found him. He was hungry and glad to be alive and all he wanted to do was to get out of the country, which he did on the first train, in spite of our urging him to stay. I don't think he ever forgave us, even if we had not invited him to come. He had written to Iowa City and when my secretary told him where Mrs. Dean and I were, he thought we were at some park.

Everyone of us know of Joe's scientific attainments and I have mentioned these incidents, hoping to give you an insight of him as a man.

You can judge a man by his intimate associates. At our medical meetings these have been the very best otolaryngologists in America.

Joe Beck secured his early education in Bohemia under the most trying circumstances. I know I would not have made much progress under those conditions. When he came to this country he could speak no English and many people not only did not help him but made things hard for him; however, he kept his gaze on a star which he placed high in the sky. He reached the star. What more can anyone do?

L. W. D.

BOOK REVIEW.

War Medicine. A Symposium. Winfield Scott Pugh, M.D., Commander (M.C.), U.S.N., Retired. Formerly Surgeon, City Hospital, New York, Editor. Edward Podolsky, M.D., Associate Editor; Dagobert D. Runes, Ph.D., Technical Editor. Five hundred and sixty-five pages with index. New York: Philosophical Library, Inc. 1942.

In the past, each war has presented its own particular medical problems, and in turn the accumulation of information obtained as the result of dealing with these problems has each time added measurably to the advancement of the sum total of medical and surgical knowledge.

The present war, because of the mechanized aspects of combat, plus the great advances made in the field of aviation, presents perhaps more of these problems than any one of the previous military conflagrations.

This book is a symposium by a selected group of American and British authors, each dealing with some particular phase of medicine or surgery as observed in the present war. Because our English allies have now had three years of experience all over the world, the contributions by our British confreres are of special importance.

To anyone interested in the medical and surgical problems arising as a result of the present war, this book is highly recommended. A. M. A.

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CONTENTS

Neurological Complications of Serum Sickness with Special Reference to the Ear. Dr. H. Marshall Taylor, Jacksonville, Fla. 923

Practical Aids in the Technique of Catheterizing, Lavaging and Injecting the Sphenoid Sinus. Dr. J. C. Peele and Dr. F. E. LeJeune, New Orleans - - - - - - - - - - - - - - - - - 933

Cricopharyngeal Spasm. Dr. Louis H. Clerf and Dr. F. J. Putney, Philadelphia - 944

Pathologic Changes of the Temporal Bone in Osteomyelitis of the Skull. Dr. Hans Brunner, Chicago - - - - - - - - - - 954

Cancer of the Esophagus: Study of 100 Consecutive Cases. Dr. Paul H. Holinger, Chicago, and Dr. H. James Hara, Hinsdale, Ill. 968

Presentation of a Simple Device for Excluding Interfering Noises from One Ear While Testing Hearing Acuity of the Other Ear. Dr. Horace Newhart, Minneapolis - - - - - - - 983

Hematology. Dr. John J. Shea, Memphis - - - - - - - - - 984

Suffocation by a Sunflower Seed. Dr. Goodlatte B. Gilmore and Dr. Maurice D. Grant, New York - - - - - - - - - - - 993

In Memoriam.

Ewing W. Day, M.D. - - - - - - - - - - - - - - - - - 995

Joseph C. Beck, M.D. - - - - - - - - - - - - - - - - - 996

Book Review - 1000